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PARAPHARYNGEAL HEMORRHAGE

DIAGNOSIS AND TREATMENT

FRANCIS W. WHITE, M.D.

AND

LOUIS HUBERT, M.D.

NEW YORK

In presenting the following cases and incidental remarks thereon, we realize that much has already been written on parapharyngeal hemorrhage. The fact remains, however, that many specialists in diseases of the ear, nose and throat have been fortunate enough not to have had any harrowing experiences with fatal or near fatal hemorrhage or fatality following septic thrombosis of the internal jugular vein. This good fortune is no guarantee for the future. To Mosher,[1] in this country, is due much of the present interest in the parapharyngeal (pharyngomaxillary) space and its inflammatory diseases. Salinger and Pearlman[2] reported 10 cases and reviewed the literature up to the date of their report. The report is of intense interest, as it concerns conditions that otolaryngologists are called on to treat. Knowledge and good judgment are required in order to give the best treatment for the patient's welfare. The more case reports there are, the sooner the symptoms, diagnosis and treatment will be clarified. A few salient points in the cases reported by Salinger and Pearlman will bear close scrutiny. There were 6 deaths, 4 before surgical aid could be instituted. There were 5 cases of ligation of the common carotid artery and 1 of the jugular vein. In 1 case the common carotid artery was ligated a second time on account of a recurring hemorrhage. These points are stressed because in the series of cases reported in this paper, some parallel experiences have been noted. Fortunately, no deaths are to be reported by us. The common carotid artery was ligated on 2 occasions, the

Read at a joint meeting of the sections on otolaryngology of the New York Academy of Medicine and the College of Physicians of Philadelphia, New York, March 16, 1938.

1. Mosher, H. P.: Deep Cervical Abscess and Thrombosis of the Internal Jugular Vein, Laryngoscope **30**:365 (June) 1920; The Submaxillary Fossa Approach to Deep Pus in the Neck, Tr. Am. Acad. Ophth. **34**:19, 1929.

2. Salinger, S., and Pearlman, S. J.: Hemorrhage from Pharyngeal and Peritonsillar Abscesses, Arch. Otolaryng. **18**:464 (Oct.) 1933.

external carotid artery on 2 and the ascending pharyngeal artery on 1. The internal jugular vein was not involved in any instance. The Horner syndrome occurred once. Peripheral paralysis of the facial nerve was observed once, but no signs of cerebral dysfunction were present in any patient. In 1 patient there were concomitant diabetes and acidosis; 1 had scarlet fever; 1 had an infection of a lower molar tooth, which was extracted, and 2 had involvement of the parotid gland. In each patient, however, some pharyngeal symptoms were present either early or late.

REPORT OF CASES

CASE 1.—J. C., a boy 5½ years old, was admitted to the Willard Parker Hospital on Jan. 1, 1937, with scarlet fever, nasopharyngitis, pansinusitis and adenotonsillitis. The right ear drum was congested and the left dull. The temperature was 101 F., the pulse rate 120 and the respiratory rate 28. There was marked cervical adenitis on the left side. On the tenth day of the disease spontaneous rupture of the left ear drum occurred. Three days later there was a bloody discharge from the left ear through the perforation. On the sixteenth day brisk bleeding from the external canal accompanied acute mastoiditis. A mastoidectomy was performed. There was excessive bleeding along the posterior wall of the canal, especially near the tip. On the seventeenth day there were profuse nosebleed and excessive bleeding from the mastoid wound. That night profuse nosebleed recurred. The attacks of nasal hemorrhage were reported to have been controlled with epinephrine packs. These hemorrhages were, undoubtedly, from the eustachian tube. Twelve days after the mastoidectomy there were epistaxis, bleeding from the mouth and much blood in the vomitus.

On the following day an operation was performed on the neck. Three large, indurated, markedly inflamed glands were removed. No pus was found. The whole region was a continuous inflammatory mass enveloping the large vessels of the neck. The jugular vein had the appearance of being about to rupture at each filling. The common carotid artery was ligated. The external and internal carotid arteries were massed in the remaining inflammatory tissue. There was a clot of blood in the immediate region of the bifurcation of the common carotid artery, which proved to be a false aneurysm. The wound was partially closed. A slight hemorrhage from the nose occurred a few hours later. The day after the operation, a sac appeared on the left lateral pharyngeal wall, extending to the region of the left arytenoid cartilage. It was punctured with a large needle, and analysis of the fluid removed by aspiration gave negative results. Massaging from below upward brought about the removal of a considerable quantity of thick blood by suction. There was no further bleeding. The patient was very ill from the time of admission and received six blood transfusions besides infusions of solution of sodium chloride. A bacteriologic examination revealed Staphylococcus aureus in the ear and Streptococcus haemolyticus in the mastoid region. Two weeks later the silk ligature was extruded through the wound in the neck. On March 23 a plastic operation was performed on the neck. The patient was discharged on April 16. The adenoids and tonsils were removed ten months later.

CASE 2.—M. S., a boy 15 years old, was admitted to the Manhattan Eye, Ear and Throat Hospital on March 13, 1935, complaining of sore throat, difficulty in opening the mouth and stiffness of the neck on the right side. Examination revealed a slight swelling on the right lateral pharyngeal wall, trismus and a temperature of 103 F. The swelling increased in size and spread over the right

anterior pillar into the soft palate. An incision into this swelling resulted in severe hemorrhages. On March 24 the right external carotid artery was ligated, but the hemorrhages continued. Two days after the first ligation, the external carotid artery was again ligated, as was the ascending pharyngeal artery. The bleeding continued, not only from the throat but from the external auditory meatus. On March 28 the wound in the neck was reopened. An incision extending from the original wound in the neck to the suprasternal notch was made, and the common carotid artery was exposed in the inferior carotid triangle and tied. There was only one more hemorrhage, on April 8, eleven days after the ligation of the common carotid artery. Since then, no more bleeding has occurred. Cultures from deep within the incisions in the pharynx and the neck showed pure Str. haemolyticus. On March 30, peripheral paralysis of the facial nerve was noticed on the right side. It was due to the spreading of the infection to the parotid space and the involvement of the facial nerve in that area. A complete Horner syndrome on the right also was observed; this has been present up to the time of writing. The paralysis of the facial nerve has entirely cleared up. The patient is in excellent condition. He was discharged on May 11.

Case 3.—J. W., a woman aged 60, was admitted to the Manhattan Eye, Ear and Throat Hospital on March 2, 1935, complaining of severe pain in the throat and neck for the previous four days. Examination revealed an enormous swelling of the right side of her neck. The swelling was most prominent below the angle at the right side of the jaw and was hard and tender. Trismus was marked. However, a swelling on the right lateral pharyngeal wall could be distinguished. Examination of the urine showed a trace of albumin, fine and coarse granular casts, blood and pus, sugar 2 per cent and a 4 plus reaction for acetone and for diacetic acid. The sugar content of the blood was 205 mg. per hundred cubic centimeters. The patient was given appropriate treatment for this condition. She was placed under local anesthesia, and the pharyngomaxillary space was explored; about 2 teaspoonfuls (8 cc.) of pus escaped. On March 30 the patient was discharged. The wound in the neck was completely healed. The urine was normal except for a faint trace of albumin. The sugar content of the blood was normal.

Case 4.—M. K., a girl aged 19, was admitted to the Manhattan Eye, Ear and Throat Hospital on Nov. 19, 1936, complaining of sore throat, stiffness and swelling of the right side of the neck. Her temperature was 103 F. The condition had started seventeen days before admission with soreness in the right side of the throat. The patient was unable to open her mouth, and deglutition was painful. There were marked trismus and a swelling on the right lateral pharyngeal wall, as well as indurated swelling with tenderness in the parotid region. The temperature ranged from 99 to 104.5 F. daily for six days, with no change in symptoms. A blood culture was negative.

On the seventh day after admission an incision was made in the right posterior pharyngeal wall. No free pus was found. Cultures taken from the depth of the wound showed Str. haemolyticus. The range of temperature was the same for the next three days. The swelling on the right side of the neck increased considerably and was especially marked below the angle of the jaw. An external incision was made. The right pharyngomaxillary space was explored. No free pus was found. A counterincision was made posterior to the primary incision. A drain was inserted and the wound closed. The temperature ranged from 99 to 103 F. for the next four days and then dropped to normal; the patient went on to recovery. She was discharged on December 17.

Case 5.—E. H., a man aged 40, was admitted to the French Hospital on Dec. 12, 1935. He was a private patient of Dr. A. M. Wright. Two days after

the extraction of a right lower molar tooth, a swelling had appeared externally at the angle of the jaw. Mastication was painful and deglutination difficult. There was trismus. Examination of the right side of the throat revealed a large peritonsillar swelling. There was marked edema of the uvula. With the patient under local anesthesia, the mass was incised and a large quantity of black blood allowed to escape. No pus could be identified. There was free drainage for about two days, but the swelling persisted several days longer. The external swelling was treated conservatively. Four days after the patient's admission, pus appeared spontaneously externally. A few days later a sequestrum in the lower alveolar process appeared and was removed from the gum. The case was one of osteomyelitis of the right side of the lower jaw, involvement of the lateral pharyngeal wall and cervical abscess. Conservative treatment was followed by recovery. The patient was discharged on Feb. 6, 1936.

CASE 6.—S. S., a man aged 47, was admitted to the Willard Parker Hospital on Jan. 29, 1937. The diagnosis on admission was parotitis. The diagnosis ultimately placed on file was parapharyngeal abscess. There were pain in the right side of the neck at the angle of the jaw, swelling in the parotid region, stiff neck and marked cervical adenitis. A small amount of pus exuded from the right tonsil. There were edema, redness and swelling from the upper level of the tonsil to the upper level of the epiglottis, which was not incised. A bacteriologic examination revealed Str. haemolyticus. The condition was treated conservatively and recovery ensued. The patient was discharged on February 3.

ANATOMIC AND DIAGNOSTIC CONSIDERATIONS

The relation of the great vessels of the neck to the lateral pharyngeal wall is of great importance. The internal carotid artery lies closer to the pharynx than does the external. The former does not diminish in size as does the latter. The internal carotid artery is much more tortuous than the external. One of the tortuosities is of an S shape and lies somewhat behind the bifurcation of the common carotid artery. Several others may be traced, one usually just before the artery enters the carotid canal and usually two after its entrance into the cranial cavity. These serve as a safety device for preventing the surging of blood into the cranial cavity and may predispose this artery to formation of a false aneurysm. The tortuosity at the bifurcation of the common carotid artery and the one just external to the entrance into the carotid canal are of importance in infections deep in the neck. Enlarged cervical glands can cause still greater slowing of the arterial flow. These glands may break down, thus bathing the neighboring structures in pus. The internal carotid artery, owing to its tortuosity, can be greatly hampered by the enlarged glands and when pus is present may become eroded and form a false aneurysm. Because of inflammatory reaction, a resulting hemorrhage is for a time confined to the immediate neighborhood. A severe hemorrhage may occur suddenly, or several small hemorrhages may precede a grave hemorrhage. Any hemorrhage from the throat, especially if repeated and if accompanying local infection, should be looked on as a danger signal, and appropriate action should be taken immediately.

The general anatomic structure of the region has been so thoroughly dealt with by other authors that it would add little to go into detail, it being sufficient to say that the integrity of the superior constrictor muscle of the pharynx is of the greatest importance. "Moreover, the superior constrictor is, at times, very thin; indeed, almost wanting, so that in essence the tonsillar capsule and the pharyngeal aponeurosis alone intervene between the attached surface of the tonsil and certain of the arteries and venous plexuses referred to."[3] Infection may pass through the muscle directly or by veins and lymphatic vessels. Besides the great blood vessels of the neck and the lymphatic glands, the pharyngomaxillary space contains the ninth, the tenth and the twelfth cranial nerve and also the cervical sympathetic trunk and its superior cervical ganglion. In the parotid space, which communicates with the pharyngomaxillary space through a gap in the fascia, is the seventh cranial nerve (fig. 1). Every effort should be made to determine whether these nerves are involved, as such knowledge may be of great aid in making a diagnosis. When the condition is doubtful, it may confirm the suspicion that pharyngomaxillary infection exists. However, such information, except in the presence of paralysis of the facial nerve, is somewhat difficult to obtain and has, in the past, been neglected because of trismus and the general condition of the patient. Therefore, one finds only a few observations on this subject in the literature.

Involvement of the Glossopharyngeal, or Ninth Cranial, Nerve.—Disturbances of this nerve are the most difficult to diagnose in the type of case under discussion. The nerve supplies the posterior third of the tongue with the sense of taste and the superior constrictor muscle with motion. Difficulty in swallowing is the most important symptom that results from loss of its function. When the tongue is protruded and the patient says "ah," a unilateral paralysis of the superior constrictor muscle is distinguishable by a lateral deviation of the pharyngeal wall toward the nonparalyzed side. This sign can be observed only early or after the pharyngeal swelling has to a great extent subsided and the trismus has greatly lessened. It is therefore of limited practical value.

Involvement of the Vagus, or Tenth Cranial, Nerve.—Hoarseness in a case in which pharyngomaxillary infection is suspected may be due to an involvement of the vagus nerve, causing a unilateral paralysis of the vocal cord. This condition is difficult to establish unless a direct examination of the larynx is undertaken. A case in which it occurred was recently observed by us. During the operative procedure on the neck, while the patient was under general anesthesia, a direct examination of the larynx revealed a paralysis of the vocal cord on the side of the pharyngomaxillary infection.

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3. Schaeffer, J. P.: Aberrant Vessels in Surgery of the Palatine and Pharyngeal Tonsils, J. A. M. A. **77**:14 (July 2) 1921.

Involvement of the Hypoglossal, or Twelfth Cranial, Nerve.—Ulrich,[4] of the Nager Clinic in Zurich, Switzerland, reviewed cases that he found reported in the literature in which a unilateral paralysis of the tongue developed in acute pharyngeal disturbances. The causal disorder was generally a severe sore throat with abscess or extensive swelling of the cervical glands. The hypoglossal paralysis was always unilateral and developed on the more severely affected side. The author also described 4 cases of his own observation. In 2 cases of fatal termination microscopic examination revealed that the lesion of the

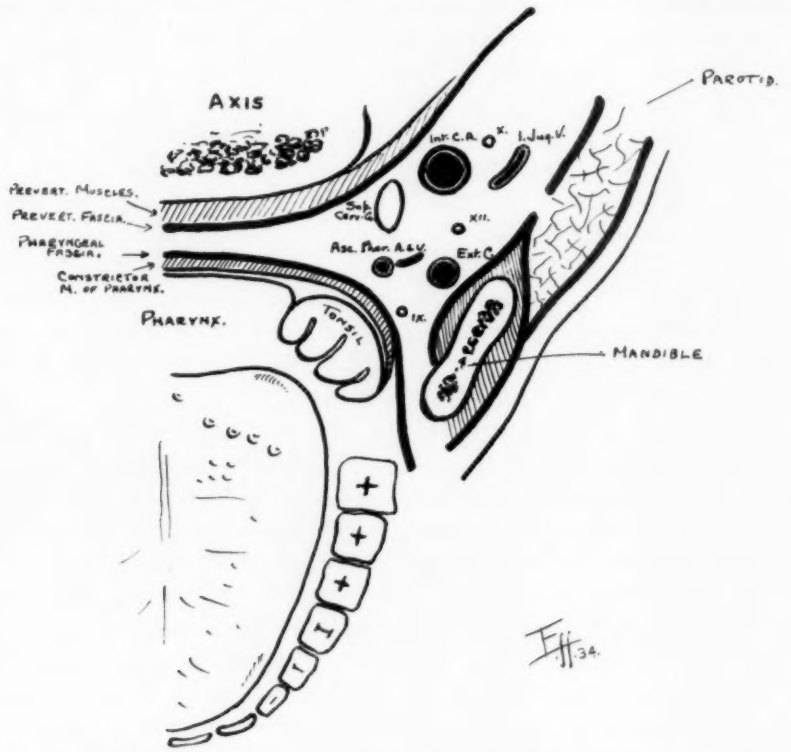

Fig. 1.—Schema of the pharyngomaxillary space. *Prevert.* signifies prevertebral; *m.*, muscle; *sup. cerv. g.*, superior cervical ganglion; *asc. phar. a. & v.*, ascending pharyngeal artery and vein; *int. c. a.*, internal carotid artery; *ext. c.*, external carotid artery; *i. jug. v.*, internal jugular vein; *IX*, *X* and *XII*, the ninth, the tenth and the twelfth cranial nerve.

hypoglossal nerve was due to periphlebitis of the internal jugular vein and of the posterior facial vein, respectively. The surgical observations in the third case, which also terminated fatally, and the clinical course in the fourth, of recovery, indicated the same mode of impairment. A

4. Ulrich, K.: Hypoglossuslähmungen bei Anginen, Schweiz. med. Wchnschr. **64**:444 (May 12) 1934.

unilateral paralysis of the tongue does not entirely paralyze this organ, because the muscles of the tongue are closely interlaced between the two sides. The tongue can be moved in all directions, though imperfectly and with difficulty. In the act of protruding the tongue, however, the healthy muscles elevate its posterior portion, pushing it outward more fully on the nonparalyzed side; hence the tongue deviates to the side of the paralysis. The paralyzed half of the tongue is atrophic, a characteristic sign.

Involvement of the Sympathetic Nervous System.—In the extension of the infection to the pharyngomaxillary space, the superior cervical ganglion and its preganglionic and postganglionic fibers may become involved, resulting in Horner's syndrome, as in 1 of our cases. This syndrome is characterized by a constriction of the pupil, a drooping of the upper eyelid, a narrowing of the palpebral fissure, an apparent or

Fig. 2.—Photograph showing dilatation of the left pupil, drooping of the lid, narrowing of the palpebral fissure, enophthalmos on the right and failure of the right pupil to dilate after instillation of a 4 per cent solution of cocaine hydrochloride.

actual sinking in of the eyeball and certain changes in the vasomotor and the sudorific activity of the skin of the face. These signs are produced by a paralysis of the sympathetic fibers that are given off by the superior cervical ganglion and supply the dilator muscle of the pupil, the smooth muscle fibers in the lid, Müller's muscle and the blood vessels and sweat glands of the skin of the face. This syndrome, although characteristic and easily diagnosed, is frequently overlooked in an acutely ill person who does not complain of any ocular symptoms. To prove the diagnosis of Horner's syndrome, a 4 per cent solution of cocaine hydrochloride is instilled into both conjunctival sacs. After fifteen to twenty minutes, the normal pupil dilates because of the stimulating effect of cocaine on the sympathetic dilator fibers of the iris; the other remains constricted (fig. 2). A case of pharyngomaxillary infection with

paralysis of the ninth and the twelfth cranial nerve and the sympathetic trunk was reported by Gaston and Lemaitre.[5] The condition started with a right peritonsillar abscess, difficulty in swallowing and high fever. Horner's syndrome developed on the right. The movements of the tongue were limited, but the local condition did not allow further neurologic examination. The peritonsillar abscess was incised and a large amount of pus evacuated. Five days later, severe hemorrhage occurred, indicating the erosion of a large blood vessel. The external carotid artery was ligated, and the bleeding stopped. In a few days, the swelling in the throat slowly disappeared, and then a more complete examination of the nerves involved was undertaken. The tongue deviated to the right, and its right half was atrophic. The right side of the soft palate was paralyzed and drawn to the left during phonation. The right half of the posterior pharyngeal wall was drawn to the left. The sense of taste was found to be diminished in the posterior fourth of the right side of the tongue, when tested by means of quinine.

PROBLEMS OF TREATMENT

Most frequently, the source of the infection is peritonsillitis. Tonsillitis or pharyngitis may also be the cause. In any event, with or without an involvement of the pharyngomaxillary vascular structures, there are fever, tenderness in the cervical region and adenitis. If the infection is of such severity as to cause erosion of a large vessel in the neighborhood, blood escapes. At this time there may be marked, although gradual, increase in the amount of swelling in the neck. If the pressure of the escaping blood is sufficient to force the neighboring tissues inward, a mass forms on the lateral wall of the pharynx and extends upward to the soft palate, simulating a peritonsillar abscess. It may even pass downward toward the base of the tongue. These swellings are most frequently diagnosed as peritonsillar or lateral pharyngeal abscesses and are incised. A varying quantity of dark blood only escapes. This result should be a danger signal of vascular damage. When a peritonsillar or lateral pharyngeal abscess has been incised, the resistance of the lateral pharyngeal wall has been greatly reduced, and a sudden severe or fatal hemorrhage may supervene. A hematoma following incision of an abscess may give rise to a seepage of blood and should be given careful consideration. Therefore, even though in many cases peritonsillar and lateral pharyngeal abscesses are cured, it behooves all otolaryngologists to be informed and ready to treat the unusual ones successfully. The necessary information can be acquired as the result of bitter experience

5. Gaston, A., and Lemaitre, Y.: Syndrôme de compression de l'espace rétro-parotidien postérieur au cours d'un phlegmon gangréneux de l'amygdale, Ann. d'oto-laryng., July 1932, p. 807.

or, better, by applying the knowledge gained by other practitioners. To this end a correlation of the facts concerning anatomic structure, causation, signs and symptoms is of the utmost importance.

The subject under discussion is infection in the neck due to causes arising primarily within the oropharyngeal cavity, involving secondarily the pharyngomaxillary space and some of the structures therein and resulting in hemorrhage into the throat, nose and ear. This paper does not deal with possible complications in the jugular vein. It is well to remember in surgical intervention in the neck that arteries have a tendency to erode and veins to become thrombosed.

Next in importance to a history of pharyngeal infection is the presence of trismus. Most frequently a peritonsillar abscess burrows along the anterior pillar in an upward direction into the soft palate. Trismus, which also is characteristic of this condition, is due to a spasm of the internal pterygoid muscle. This spasm, however, is not due to an actual infection of the space but to collateral edema. Trismus is conspicuous by its absence in cases in which peritonsillar abscess burrows down the posterior pillar and forms a mass on the lateral pharyngeal wall. Cases of this type are not as frequent as those in which trismus is present. If a supposed anterior peritonsillar abscess is incised and trismus is not favorably affected, it must be assumed that pharyngomaxillary involvement is present. If no pus but dark, bloody fluid appears, the patient should be hospitalized. If a slight amount of bleeding continues after incision, a differential diagnosis of superficial or deep bleeding must be made. If the bleeding is from the edges of the incision, it may be controlled by local applications. Continued bleeding then indicates that deeper structures are involved, and ligations of the external carotid and of the ascending pharyngeal artery should be done and a loose ligature placed about the common carotid artery for use in case of necessity, when fatal hemorrhage may thus be forestalled. While fatal hemorrhage occurs comparatively infrequently, still it occurs too frequently. Many patients die from unannounced, sudden, overwhelming but avoidable hemorrhages. A hemorrhage from the external auditory canal or the eustachian tube practically always means erosion of the internal carotid artery and the formation of a false aneurysm. It is an indication for no temporizing but quick and definite decision, to ligate the common carotid artery. As noted in the literature,[2] about 80 per cent of patients with such a hemorrhage have died. Ligation of the common carotid artery in all cases gives at least a 2 to 1 chance. The ligation should be done slowly. The idea is to stop the flow of blood through the artery, not just to cause a solution of continuity of the intima of the artery. This is the crux of ligation of the common carotid artery! While the foregoing procedure may sound drastic, emergency

life-saving surgical intervention is not in the field of cosmetic surgery. The plastic surgeon can be depended on to give more joy than the mortician!

Associated with hemorrhage from the throat or ear, pus may be formed deep in the neck. The patient is then threatened not only by fatal hemorrhage but by acute and lethal edema of the larynx. Furthermore, rupture of the pus into the larynx or into the trachea may suddenly occur and in either case prove fatal. The necessity of a clearcut diagnosis of pharyngomaxillary infection is obvious. Trying to lessen the lateral pharyngeal swelling by incising it is worse than futile. The external route of attack is the only one to be considered. One may operate with the patient under local or under general anesthesia. If general anesthesia is to be employed, the intratracheal method is the safest. By this method, complete packing off of the pharynx is possible, and any sudden hemorrhage or rupture of pus into the upper airway can easily be handled by suction. Before the intratracheal tube is introduced, a clear view of the larynx and the upper part of the trachea may be obtained by direct inspection. It may be surprising to find involvement of the vagus nerve, which originates in the pharyngomaxillary fossa. From what has been said, it is evident that in the absence of definite inflammatory involvement of the tonsillar area no incision into a lateral pharyngeal mass should be made. It is better to err on the side of safety by exploring through an external incision. Naturally, the condition of the internal jugular vein can be determined at the same time.

CONCLUSIONS

An analysis of reported cases of pharyngomaxillary infection suggests the following:

1. The degree of infectivity of the causative organisms for a given person determines the seriousness of the pharyngomaxillary invasion.

2. Conservative treatment of pharyngomaxillary infection is in many instances successful.

3. An effort should be made to determine the status of the nerves in the pharyngomaxillary space.

4. Free, active hemorrhage from the throat, with or without apparent inflammation, requires not local treatment but bold external operative measures.

5. Meddlesome incision of a lateral pharyngeal swelling may lead to hemorrhage. If the condition is treated conservatively, ligation may not be necessary.

6. Aural hemorrhage associated with pharyngomaxillary infection requires immediate ligation of the common carotid artery.

7. If any doubt exists regarding erosion of the internal carotid artery, the external carotid and the ascending pharyngeal artery should be ligated and a loose ligature placed around the common carotid artery.

8. Ligation of the common carotid artery, when it is done slowly —in minutes, not seconds— and an attempt is made actually to prevent the flow of blood beyond the ligature, not just to feel the severance of the intima, is a life-saving operation, as compared to temporizing methods of operation by the intrapharyngeal route.

9. Hemorrhages from the throat and the ear associated with trismus, involvement of nerves as noted, lateral pharyngeal tumefaction, sepsis and swelling and tenderness of the corresponding side of the neck indicate infection of the pharyngomaxillary fossa.

471 Park Avenue.

161 East Seventy-Ninth Street.

RELATION OF THE INTERNAL EAR SPACES TO THE MENINGES

H. B. PERLMAN, M.D.
AND
J. R. LINDSAY, M.D.
CHICAGO

The pathologic picture of meningitis, particularly in its relation to that of the temporal bone, is a matter of particular concern to the otologist, since in a large percentage of cases meningitis develops from otitic infection. Some of the main questions which arise in an investigation of the subject are (1) by what various pathways and methods infection may extend from the temporal bone to the meninges and intracranial structures and (2) what relation labyrinthine infection bears both to suppuration in the middle ear spaces and to infection within the subarachnoid space. Information regarding the latter point, namely, the relation of labyrinthitis to infection of the subarachnoid space, may be obtained from a study of cases in which an early stage of extension to the labyrinth from the meninges is exhibited. Observation of such cases and of cases of subarachnoid hemorrhage and intracranial tumor affords information regarding the anatomic and physiologic relation of the fluids within the labyrinth and the subarachnoid space.

The late stages of labyrinthitis following meningitis, as found in deaf mutes, have been described by Siebenmann,[1] Panse[2] and others.[3] Extensive studies of labyrinthitis secondary to disease of the middle ear, including histologic study of the temporal bone, have been made by Fraser and Turner[4] and by Dean and Wolff.[5] The more acute process in the early stages of infection has received considerable attention, both

From the Department of Surgery, Division of Otolaryngology, University of Chicago.

1. Siebenmann, F.: Grundzüge der Anatomie und Pathogenese der Taubstummheit, Wiesbaden, J. F. Bergmann, 1904, vol. 1.

2. Panse, R. E. O.: Pathologische Anatomie des Ohres, Leipzig, F. C. W. Vogel, 1912.

3. Brikholz, H., in Denker, A., and Kahler, O.: Handbuch der Hals-, Nasen- und Ohrenheilkunde, Berlin, Julius Springer, 1926, vol. 6, p. 766.

4. Fraser, J. S., and Turner, A. L.: J. Laryng. & Otol. **43**:609, 1928.

5. Dean, L. W., and Wolff, D.: Ann. Otol., Rhin. & Laryng. **43**:702, 1934.

clinically and experimentally, in Europe, while in this country pertinent observations have been reported by Crowe.[6]

The study of material that has become available in our laboratory brought out a number of problems which appear to warrant reexamination and emphasis and the solution of which would aid one in obtaining a better understanding of the relation between meningitis and disease of the temporal bone.

Additional information has been provided also concerning the ramifications of the arachnoid in the temporal bone and the relation of the perilymph to the cerebrospinal fluid.

In a study of the early phases of inflammation, before a massive invasion of the labyrinth by inflammatory cells and necrosis takes place, one may observe the distribution of small numbers of inflammatory cells in their relation to the cochlear aqueduct and to the nerves in the internal acoustic meatus. Similarily one may study the distribution of red blood cells and tumor cells in the subarachnoid space. In this way observations can be made that equal in value those resulting from animal experimentation in which a record is made of the distribution of particulate matter in the temporal bone after its injection into the subarachnoid space.

The historical development of knowledge of relations of the subarachnoid space with the labyrinth has been briefly and clearly set forth by Jampolsky,[7] who has shown, moreover, in his experimental work that it is essential to study these problems in the living and that studies of the effects of injections carried out after death lead to erroneous conclusions. Further difficulties in applying the results of animal experimentation to man appear when one considers the comparative anatomy of the labyrinth. For example, the cochlear aqueduct is much longer and narrower in man than in the dog, cat or rabbit, while the modiolar spaces in the human being are larger than in the experimental animal. In the experimental animal the structures within the modiolus are separated from the scala tympani and scala vestibuli by a thin layer of compact bone, while in the human being part of this layer of bone is replaced by loose areolar tissue (fig. 1).

Crowe [6] studied the distribution of red blood cells in cases of subarachnoid hemorrhage and concluded that there are at least five routes by which the temporal bone may be invaded by blood cells:

1. Through the sheath of the facial nerve as far as the geniculate ganglion.

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6. Crowe, S. J.: Pathologic Changes in Meningitis of the Internal Ear, Arch. Otolaryng. **11**:537 (May) 1930.

7. Jampolsky, L. N.: Ann. d'oto-laryng., March 1934, p. 264.

2. Through the various branches of the vestibular nerve as far as their end organs.

3. Along the course of the cochlear nerve at least as far as the spiral ganglion.

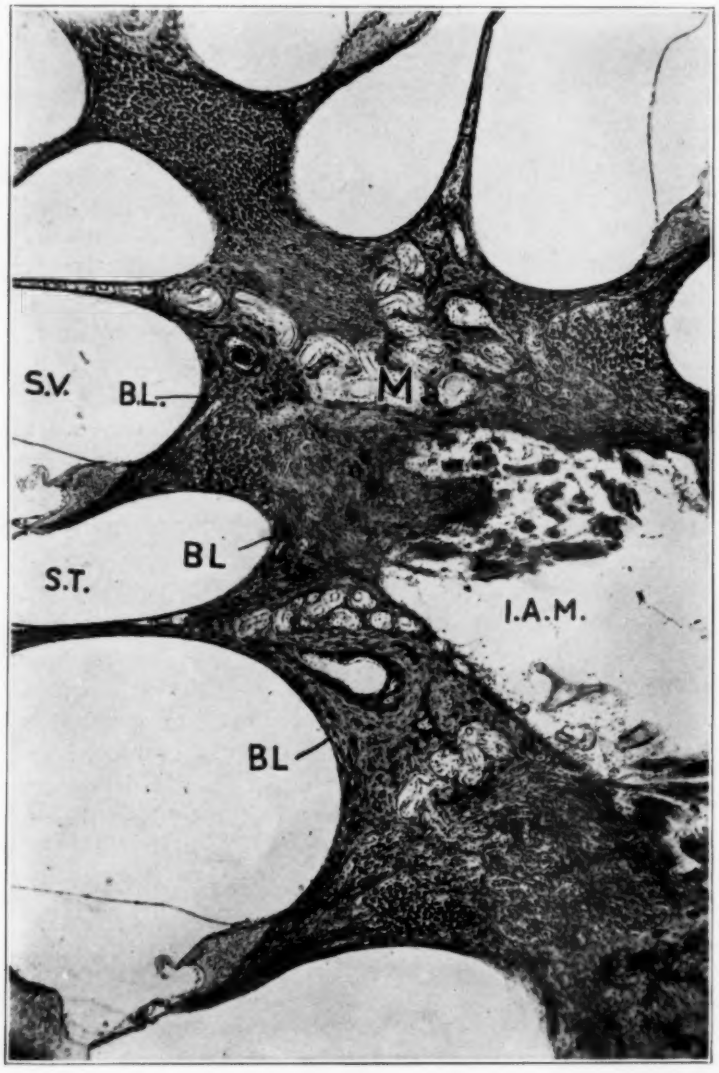

Fig. 1.—Photomicrograph (× 52.5) of a section of the ear of a cat, showing the bony laminas (*B.L.*) which separate the perilymphatic fluid in the scala tympani (*S.T.*) and scala vestibuli (*S.V.*) from the structures within the modiolus (*M.*); *I.A.M.* indicates the internal acoustic meatus. The corresponding area in the human being is shown in figure 4.

4. Along the course of the blood vessels into the cochlea.

5. Along the cochlear aqueduct.

In our material, comprising 58 temporal bones from 30 patients who had died of meningitis in acute stages, the earliest manifestation of invasion of the labyrinth that was observed consists of a collection of inflammatory cells in the subarachnoid space about the seventh and the eighth cranial nerve in the internal acoustic meatus, without destruction of the arachnoid (fig. 2). Jacobsgaard [8] observed that the distribution

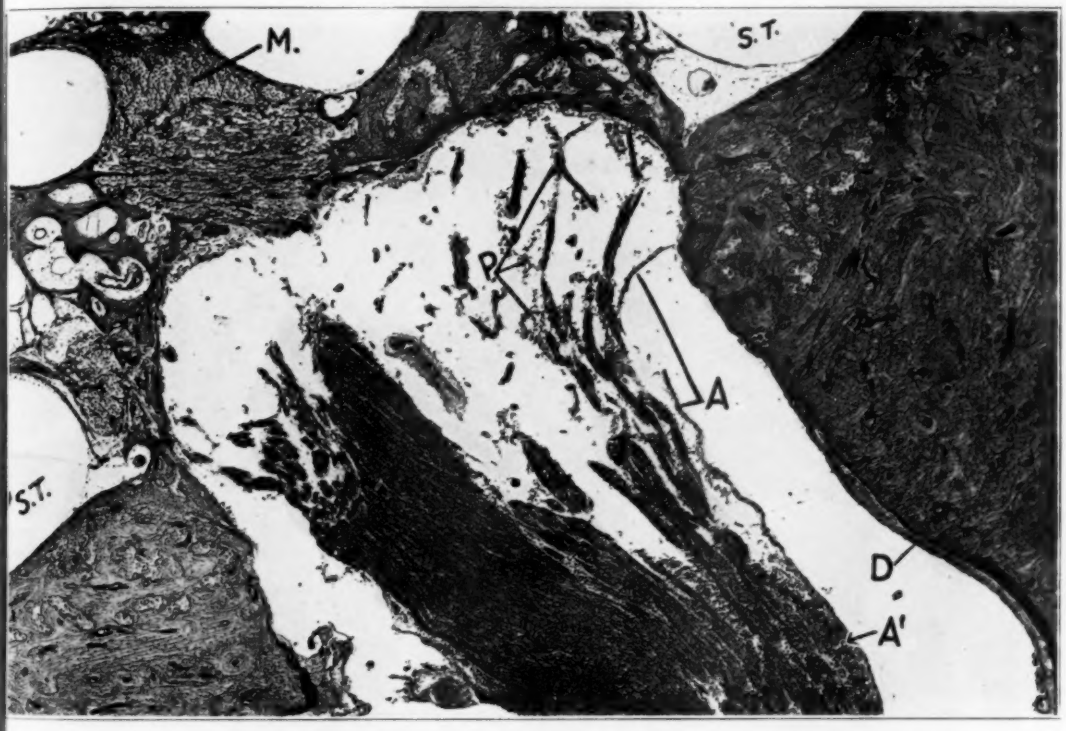

Fig. 2.—Photomicrograph (× 22.2) of a section of a human ear, showing the early stage of meningitis in the subarachnoid space in the internal acoustic meatus, without invasion of the modiolus or perilymphatic spaces of the labyrinth; *A.* is the arachnoid, under which at *A'.* is a dense collection of pus cells. Pus cells (*P.*) are scattered between the fibers of the cochlear nerve; *M.*, the modiolus, and *S.T.*, the scala tympani of the basal coil, are both free of pus cells; *D* indicates the dura.

of pus along the olfactory nerves in cases of meningitis extends to the cribriform plate. A demonstrable subarachnoid space can usually be traced to the mouth of the foramen for the facial nerve in the internal acoustic meatus, sometimes as far as the geniculate ganglion and rarely

8. Jacobsgaard, J.: Ztschr. f. Laryng., Rhin., Otol. **26**:387, 1935.

beyond it, and pus cells in the subarachnoid space about the facial nerve in this area can be observed. Usually just beyond the foramen the pia-arachnoid becomes closely applied to the trunk of the facial nerve to form its perineurium, effecting a barrier to pus cells extending along the facial canal in the early stages of infection.

There is a similar distribution of the arachnoid about the branches of the eighth nerve. At the foramens in the internal auditory meatus the arachnoid and the dura unite, and the arachnoid continues as the perineum, obliterating the subdural and subarachnoid spaces.

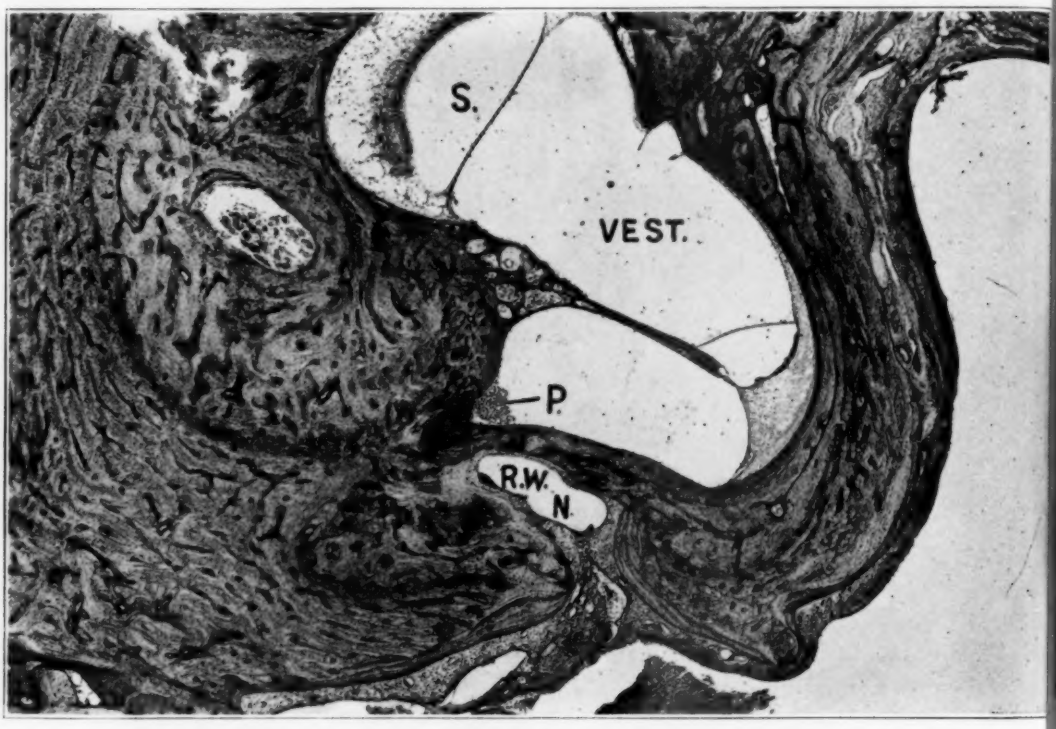

Fig. 3.—Photomicrograph (× 13.8) of a section through the left ear, showing the collection of pus (*P.*) at the mouth of the cochlear aqueduct in the scala tympani; *S.* indicates the saccule, *VEST.* the vestibule of the labyrinth and *R.W.N.* the round window niche.

In these early stages of meningitis the fluid in the perilymphatic spaces exhibits an increased affinity for the eosin stain, which indicates that early chemical changes have taken place. This reaction makes its appearance first in the scala tympani adjacent to the round window and in later stages involves the perilymphatic spaces more generally, at which time they stand out in contrast to the endolymphatic spaces.

The next degree of inflammation noted in our material is an invasion of the cochlear aqueduct and the appearance of pus cells about its opening into the scala tympani, without destruction of the arachnoid meshwork in the aqueduct (fig. 3). At this stage there are a few pus cells in the modiolar spaces and about and within the branches of the eighth nerve as they proceed from the internal meatus through the temporal bone to their end organs. There is no special concentration of pus cells in relation either to the spiral ganglion or to any particular part of the auditory nerve as it ramifies through the lamina cribrosa.

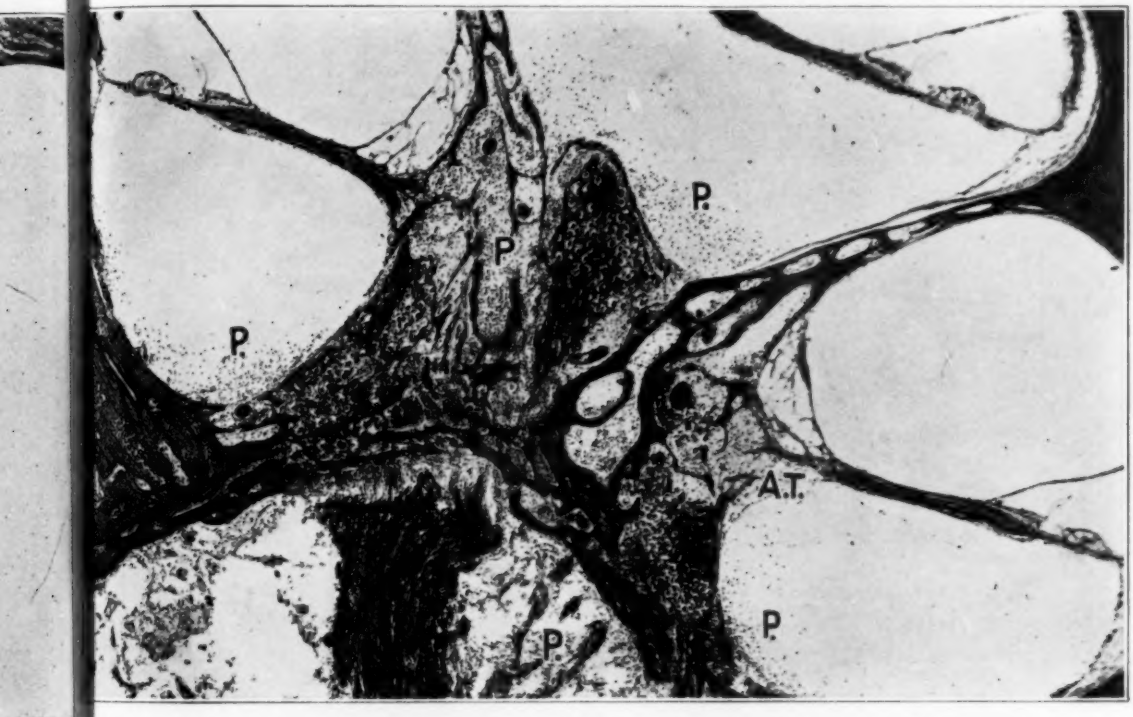

Fig. 4.—Photomicrograph (× 27.7) of a section through the modiolus in a case of meningitis, showing the early stages of extension of pus cells (*P.*) from the subarachnoid space in the internal acoustic meatus through the modiolus into the adjacent perilymphatic spaces; *A.T.* is loose areolar tissue of the modiolus adjacent to the perilymphatic space, which is in contrast to the bony lamina in the common experimental animal (fig. 1).

Although the subarachnoid space in the internal auditory meatus may contain a large collection of pus cells, the extension of the pus is at first limited at the lamina cribrosa, where the subarachnoid space ends. However, within a short time diffuse invasion of pus cells along the various branches of the nerves and vessels going through the lamina cribrosa and eventually reaching the perilymphatic space is common.

The extension of pus cells from the subarachnoid space into the modiolar spaces may either represent diapedesis through covering membranes of the nerves and vessels or follow destruction of the pia at the point of exit of these structures. That this process of invasion through the modiolus may occur in a short time after the onset of meningitis has been demonstrated in a case in which rupture of an abscess of the brain at operation was followed by meningitis and death in twenty-four hours.

Fig. 5.—Photomicrograph (× 18) of a section through the temporal bone in the region of the internal acoustic meatus, showing an avenue of extension of infection from within the temporal bone along vascular spaces (*V.S.*), with formation of new bone against the cortex in the internal acoustic meatus (*N.B.*) and inflammatory thickening of the dura (*D.*) Large masses of pus cells (*P.*) in the subarachnoid space are confined by the arachnoid (*A.*); *Ad.* indicates an adhesion between the arachnoid and the dura, *M.S.* the saccular macula and *S.G.* Scarpa's ganglion.

The next degree of inflammatory change of the labyrinth in meningitis is marked by the presence of pus cells in the perilymphatic spaces. The scala tympani of the basal coil adjacent to the inferior cochlear vein is invaded by pus cells at an early stage. In a still later stage pus cells are seen in the scala tympani and scala vestibuli of all coils, concen-

trated particularly in that part of the perilymphatic space adjacent to the modiolus (fig. 4).

As the infection in the subarachnoid space advances, adhesions between the arachnoid and the dura develop; a spread of infection to the subdural space may occur, and the dura may become thickened and infiltrated with inflammatory cells. The infection spreads along the facial canal to the limits of the subarachnoid space. When the subarachnoid space extends as far as the ganglion, it is probable that

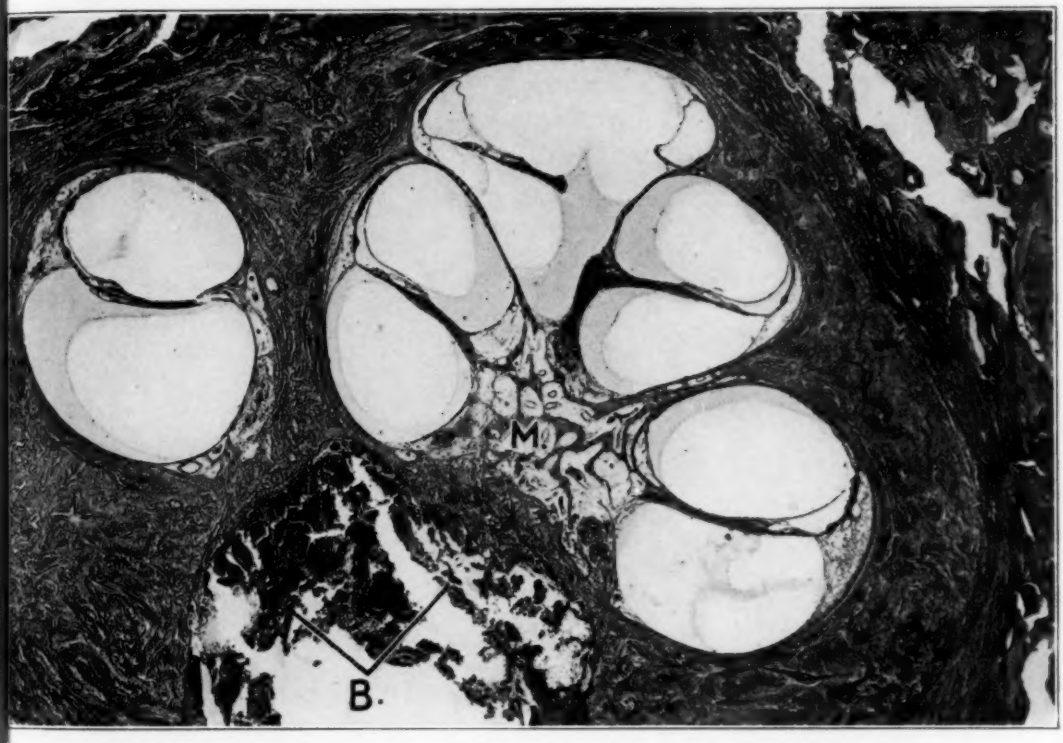

Fig. 6.—Photomicrograph (× 18.7) of a section through the cochlea in a case of subarachnoid hemorrhage, showing (*B.*) a collection of blood in the internal acoustic meatus, limited at the lamina cribrosa. The spaces within the cochlea and the modiolus (*M.*) have not been invaded.

swelling and edema accompanying the infection will be sufficient to produce palsy of the facial nerve. In some cases pus cells have been seen to extend from the subarachnoid space through the pia-arachnoid along the nerve trunk as far as the geniculate ganglion. Simultaneously the labyrinth is massively invaded by inflammatory cells, with necrosis, although the cochlear duct and the other endolymphatic spaces are last to succumb. Every patient examined, regardless of the duration of the

disease, showed pathologic evidence of meningitis in the region of the internal acoustic meatus. Almost all showed invasion of the modiolus and cochlear aqueduct. In the early stages of diffuse meningitis, which occur in most cases less than a week after the onset of the disease, formation of new bone is not seen in the labyrinth or in the cribriform area of the internal acoustic meatus. In cases of otitic meningitis, however, it is not uncommon to observe a layer of new bone outside the dura, either in the internal auditory meatus or elsewhere on the dural

Fig. 7.—Photomicrograph (× 10.4) of a section through the temporal bone in a case of meningeal sarcoma, showing (*T.*) the tumor filling the internal acoustic meatus but not invading the modiolus or the supporting tissue of the saccule (*S.*); *U.* indicates the utricle, *St.* the footplate of the stapes and *O.* a focus of otosclerosis.

surface of the bone, which has developed as a result of extension of infection and absorption of bone along vascular channels, through the cortex, from a focus of suppuration in the temporal bone. Such a manifestation represents one phase or stage of the extension of suppuration within the bone to the intracranial structures (fig. 5).

The observations on the early localization of inflammatory cells as well as red blood cells (fig. 6) and tumor cells (fig. 7) to the subarach-

noid space in the internal acoustic meatus suggest that there is no direct continuity of the subarachnoid space in this region with the structures without the modiolus and thus no continuity of cerebrospinal fluid with the perilymphatic fluid through the modiolus.

Conclusions regarding the detailed histologic structure of the root of the cranial nerve in relation to the adjacent part of the brain and its coverings could not be made from our material. As a result of his recent studies, in which he used special staining technic, Tarlov [9]

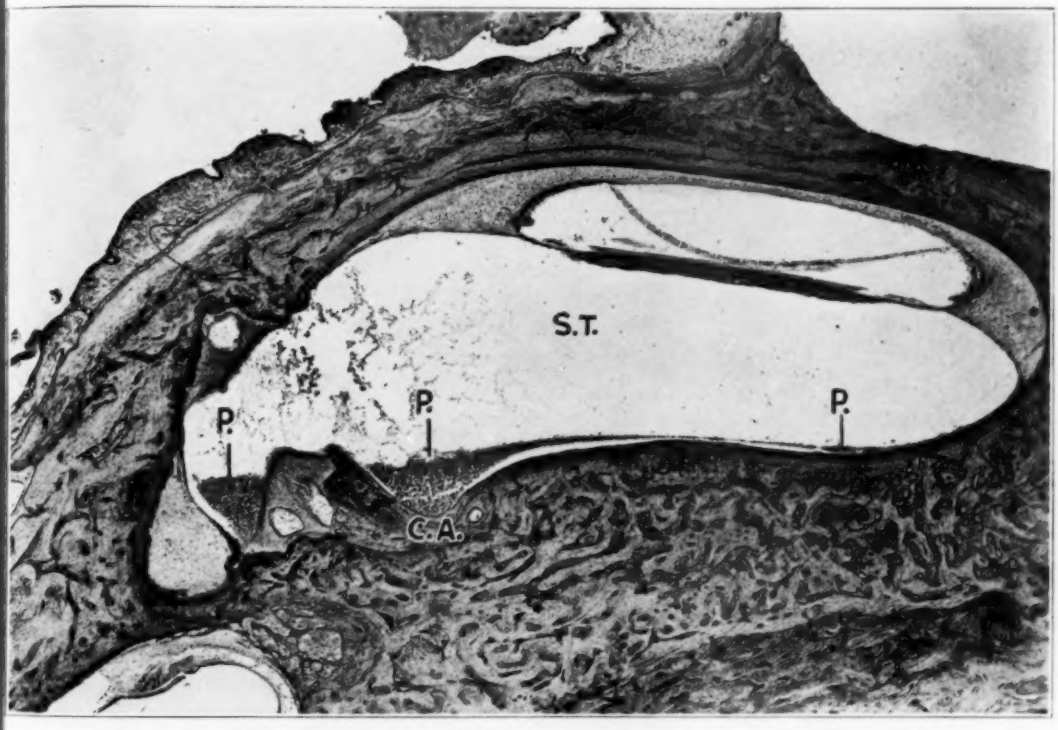

Fig. 8.—Photomicrograph (× 12.5) through the basal coil of the left cochlea in a case of meningitis, showing pus cells (*P.*) collected densely at the mouth of the cochlear aqueduct (*C.A.*) and extending up along the scala tympani (*S.T.*) in diminishing numbers. The upper coils of the scala tympani, as well as the scala vestibuli and the cochlear duct, contained no pus cells.

recorded these relations for the eighth cranial nerve as well as for other cranial nerves.

9. Tarlov, I. M.: Structure of the Nerve Root: Nature of the Junction Between the Central and the Peripheral Nervous System, Arch. Neurol. & Psychiat. **37**:555 (March) 1937.

In the living dog Jampolsky [10] observed dialyzed particles of ink in all the modiolar spaces and in the maculas in the vestibule, as well as in the cochlear aqueduct and the scala tympani of the basal coil, after injection into the subarachnoid space. In only 1 instance, and then only after repeated injections over a number of months, did he find any particles of ink above the scala tympani of the basal coil. This observation agreed well with our finding in cases of meningitis of a corresponding distribution of pus cells in the cochlear aqueduct and in the scala tympani

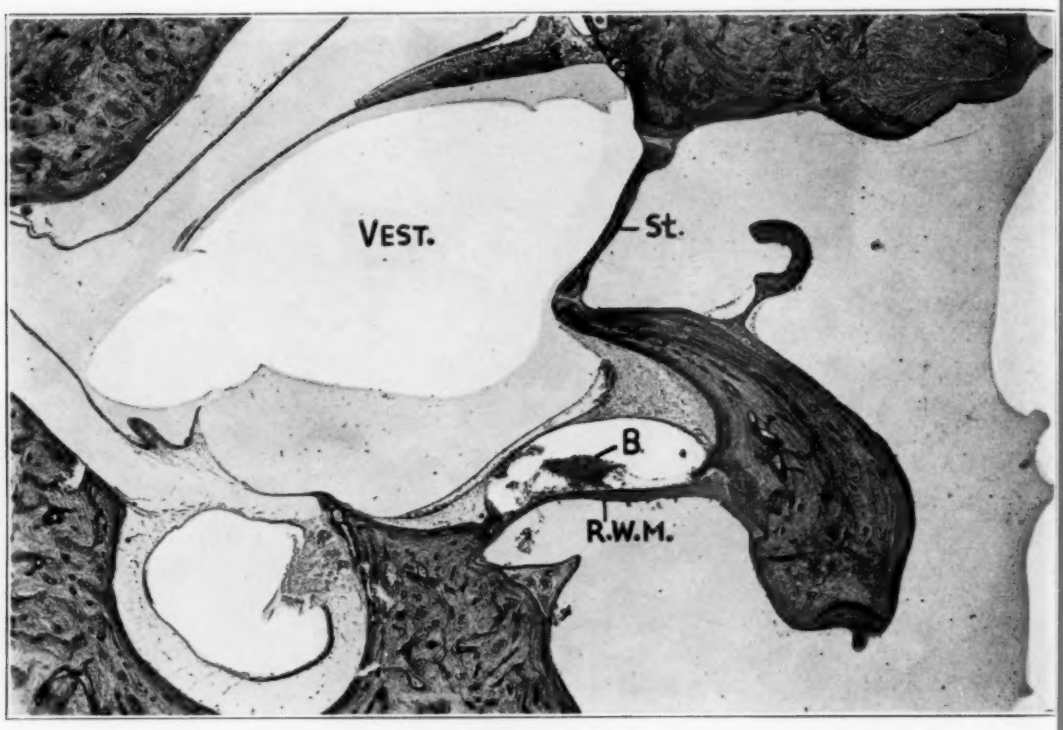

Fig. 9.—Photomicrograph (× 12.5) of a section of the left ear in a case of subarachnoid hemorrhage, showing blood (*B.*) in the scala tympani against the round window membrane (*R.W.M.*) near the opening of the cochlear aqueduct; *VEST.* indicates the vestibule and *St.* the foot plate of the stapes.

about its opening. However, conclusions regarding movements of the perilymph from these observations are difficult, since many factors must be considered. There are, for example, variations in the length and diameter of the cochlear aqueduct and in the normal pressure of the cerebrospinal fluid at the level of the internal acoustic meatuses in the human being when reclining and when erect as compared to the experimental animal, as well as variations in the pressure of the cerebro-

10. Jampolsky, L. N.: Monatschr. f. Ohrenh. **69**:23, 1935.

spinal fluid consequent to disease. Patients with meningitis are confined to the horizontal position, and the pressure of the cerebrospinal fluid is usually abnormally high. Also, it may be suggested at this point that the extremely long narrow cochlear aqueduct of man, in contrast to that of the quadruped—the rabbit, for example—may be a means by which disturbance of the perilymphatic fluid is prevented during change of posture. It is probable that any procedure to reduce or obliterate the subarachnoid space about the cranial nerves and brain stem would assist in preventing the spread of infection along these nerves and thereby the subsequent spread into the labyrinth. Such an effect is probably produced by frequent or continuous drainage of the spinal fluid or drainage of the subarachnoid cisternae.

In 3 of our specimens pus cells in diminishing numbers could be seen scattered along the wall of the scala tympani of the basal coil leading away from the mouth of the cochlear aqueduct, where there was a dense mass of cells (fig. 8).

The patency of the cochlear aqueduct was further shown in cases of subarachnoid hemorrhage, in which we often saw red blood cells in the scala tympani at the mouth of the aqueduct (fig. 9). These observations suggest that if any fluid movement occurs between perilymph and cerebrospinal fluid it is slow and probably progresses from the subarachnoid space toward the cochlea. A further observation which seemed to corroborate this opinion was that in cases in which the cochlea had been experimentally injured so as to produce bleeding within the perilymphatic spaces there was no tendency for the blood cells to collect in the region of the cochlear aqueduct. The collections of blood cells were more likely to be seen in the coils of the cochlea nearest the site of injury. These findings point to the absence of any circulation of perilymph within the cochlea. Crowe [6] observed a collection of red blood cells in the scala tympani in many cases in which operation for brain tumor had recently been performed and pointed out the possible clinical significance of such an accumulation of blood in cases of injury to the head in which no direct injury to the temporal bone could be demonstrated. He suggested that the irritative effect of the blood in this region on the arachnoid in the aqueduct and on the end organ may result in deafness. On the other hand, it should be recorded that in our clinic a large number of children have undergone craniotomy for tumor of the posterior fossa without suffering noticeable impairment of hearing and that the hearing of a number of adults who have undergone craniotomy has been found unimpaired.

The pathologic material studied has afforded some evidence on the physiologic relations between the cerebrospinal fluid and the fluids within the labyrinth. The practical application of this information may not be at once apparent, but its possible bearing on certain obscure clinical problems relating to the inner ear must be recognized.

OSTEOMYELITIS OF THE SKULL COMPLICATING MASTOIDITIS AND FRONTAL SINUSITIS

REPORT OF TWO CASES

OTTO C. HIRST, M.D.
PHILADELPHIA

Progressive osteomyelitis of the skull originating in the temporal bone is rare, and comparatively few cases have been reported. Tobey, in a review of the literature in 1926, gave disease of the ear as the etiologic factor in 53 of the 61 cases reported, with a mortality rate of 50 per cent. He further stated that the mortality rate is invariably higher in cases in which conservative treatment is employed. Because of the meager personal experience available, the physician is dependent on the literature for guidance in the treatment of this unfortunate complication. I therefore record observations, symptoms and signs from my experience with 2 cases, in the hope that the decision as to the management of my next case of progressive osteomyelitis of the skull and the conclusions on which I base that decision may be of help to others.

In analyzing the records of published cases, I find that this tragic sequel to mastoiditis or frontal sinusitis is reported to have occurred in cases in which there was no surgical intervention and in others in which it was thought that there was too little or too much surgical intervention. Because of this confusion of opinions as regards causation and as a result of the experience gained in my own cases, I am inclined to agree with Dan McKenzie that the origin of the condition lies hidden in the nature of certain otic and sinal suppurations and that its development is quite beyond control, except perhaps by avoidance of too early operation. Kopetzky stated that in cases of chronic mastoiditis thrombophlebitic changes in the vessels of the mucous membranes, due to the infection, will produce lesions in the directly adjacent bone. Retention of pus then acts as an irritant, with resulting caries and necrosis of the osseous structures. Microscopic examination will show the lesion to be of lacunar type, with osteoclasts and pus cells in the channels of the bone marrow. These structural changes must be regarded as localized pathologic osteomyelitis. Such hidden localized osteomyelitis may

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From the Hospital of the Protestant Episcopal Church, Otolaryngological Service "B," and the Graduate School of Medicine and Graduate Hospital, University of Pennsylvania, Otorhinologic Service of Dr. George M. Coates.

become acute and spread through the meningeal veins, the diploic veins and the channels of Breschet, as is so well demonstrated in Batson's specimens.

According to Zuckerkandl, 20 per cent of mastoid tips are of the diploic type, and, as the diploe contains bone marrow, one may reasonably assume that localized osteomyelitis is present in about 20 per cent of cases of acute mastoiditis. Wilensky mentioned two reasons why this condition does not more frequently follow mastoiditis. To begin with, he emphasized the fact that the pneumatic portion of the temporal bone, which makes up the mastoid, is surrounded in the superior, anterior and posterior directions by a continuous area in which diploe between the tables of the skull is at a minimum. Secondly, he called attention to the fact that the aim of operation is to remove all of the pneumatic portion. When the operation is efficiently done, the entire cavity should be bounded by dense bone, which constitutes a fairly efficient, though not constant, barrier to the transfer of infection into the cranial diploe.

Boyd-Snee stated that the signs and symptoms of progressive osteomyelitis of the skull are variable, corresponding with any complications which may supervene, as is most clearly illustrated by the progression of events in my first case. I feel that the signs and symptoms of otitic osteomyelitis of the skull may be conveniently divided into two groups: a group of early symptoms, becoming manifest in the period during which the temporal bone is involved, and a group of later symptoms, developing as the other bones of the calvarium become affected. The early signs noted included drooping of the superior wall of the canal, facial paralysis, vestibular disturbances and disturbances of the temporomaxillary joint. With symptoms of toxemia, a constant late symptom was pain in the upper molar teeth of the affected side.

REPORT OF CASES

CASE 1.—Mrs. K. S., referred by Dr. Howard Heinkel, was first seen in September 1932. At that time my diagnosis was chronic tonsillitis, a deflected septum, chronic catarrhal otitis media and chronic suppurative otitis media on the right with aural polypi. Tonsillectomy and radical mastoidectomy were advised but refused. The patient was admitted to the hospital four times for five major and six minor operations during a total period of observation of fourteen months.

After removal of the aural polypi and treatment with iodine-boric acid powder, her condition improved, but she was admitted to the hospital nine months later, on June 20, 1933, for treatment of acute mastoiditis on the right, superimposed on the chronic process.

A study of the patient's medical history revealed that she had suffered an injury to her spine at the age of 5 years, which was followed by spinal deformity. Her two children were delivered by cesarean section. Physical examination on June 20, 1933, showed her condition to be essentially normal, except for the aural discharge on the right, with swelling and tenderness over the mastoid process of the same side and marked, probably tuberculous, kyphosis of the lower dorsal region. Her

weight was 65 pounds (29.5 Kg.) and her height 4½ feet (137 cm.). The Wassermann test, tests for sugar in the blood and other routine laboratory tests were negative. The leukocyte count was 10,560. Roentgen examination at this time showed chronic, sclerosing mastoiditis on the right side.

First Operation and Postoperative Course.—On June 20 a radical mastoidectomy was performed. Convalescence was uneventful, and the patient left the hospital on June 28. Discharge from the right ear ceased after six weeks. Streptococcus haemolyticus in pure culture was reported from cultures made at the time of operation.

Three weeks after the radical mastoidectomy there was noted a marked drooping of the superior anterior wall of the canal and later of the remaining posterior wall, which I believe to have been the first sign of osteomyelitis, secondary to a periosteomyelitic swelling of the bone in the region of the zygoma. Boyd-Snee likewise noted this as the first sign of osteomyelitis in his cases.

Seven weeks after the radical mastoidectomy, the patient complained of occasional headache, nausea and projectile vomiting. She had no fever. During the eighth week she suffered continuous headache, nausea and vomiting, and a temperature of 102 F. suddenly developed, with right-sided facial paralysis. There was no vertigo. Facial paralysis appeared as the second sign of osteomyelitis of the temporal bone and was, in my opinion, due to swelling of the facial canal and pressure on the nerve.

On readmission to the hospital, on August 29, roentgen examination of the right mastoid revealed the operative defect, with considerably increased density of the cells in the region of the antrum and of those extending forward toward the zygoma. On the left side this area was well pneumatized and appeared normal. On the affected side the findings were indicative of a chronic inflammatory process producing sclerosis in this region. The tegmen tympani appeared intact. An absence of cellular detail was noted in the outer half of the petrous bone, with areas of decreased density alternating with areas of increased density, apparently due to an old operative procedure and sclerosis. There was no demonstrable erosion of the upper border. The pineal gland was calcified and showed no shift (fig. 1). An audiogram taken at this time showed a loss of hearing of 60 per cent in the right ear and 14 per cent in the left ear. Bárány's test suggested a lesion of the posterior fossa, probably abscess of the right cerebellar lobe, an assumption later proved incorrect. Unusual disturbances of the vestibular apparatus are to be expected in osteomyelitis of the temporal bone.

Eleven days later, on September 9, the right ear again began to discharge, and the facial paralysis gradually disappeared. At this time an interesting symptom of osteomyelitis of the skull appeared, namely, pain in apparently normal upper molar teeth on the affected side. I attributed this to neuralgia of the superior maxillary division of the fifth nerve, due to irritation of its meningeal branch. This symptom recurred simultaneously with the onset of each new osteomyelitic involvement. Lillie, Auriti, Kopetzky and J. Gordon Wilson have noted neuralgia and other disturbances of the fifth, or trigeminus, nerve coincident with inflammation of the mastoid and the middle ear.

My treatment at this time was expectant and symptomatic, owing to the fact that since the patient's admission pneumothorax of probably tuberculous or metastatic origin had developed on the left. On October 12 the pain in her teeth became so intense that she left the hospital to receive treatment from her own dentist. At this time her ear was still discharging, and she had a temperature ranging from normal to 100 F. The facial paralysis had disappeared.

Two weeks later and four months after the radical mastoidectomy, she was again admitted to the hospital, complaining of pain in the ear and about the right side of the upper jaw. Laboratory findings were normal.

Second Operation and Postoperative Course.—On revision, on October 27, the sclerotic bone left in the mastoid tip at the first operation was found softened and was completely removed. The base of the pyramid was examined for fistula; the middle and the posterior fossa were exposed and examined for abscess, as suggested by the findings in Bárány's test; the results were negative, and the wound was closed by another Panse flap. The patient was discharged in an improved

Fig. 1 (case 1).—Roentgenogram made on Aug. 29, 1933.

condition on November 8, her temperature at this time ranging from normal to 99 F. Three weeks later and about six months after her acute mastoiditis, on December 1, I noticed for the first time one of the later signs of progressive otitic osteomyelitis, i. e., a swelling of the right side of the head over the squamous portion of the temporal bone, extending to the zygomatic portion of the malar bone. As described in textbooks, it was doughy to the touch and tender. In conjunction with it she suffered right-sided headache, pain in her right upper molars and inability fully to open her mouth. According to Costen, disturbances of the temporomandibular joint may be expected with lesions of the temporal bone.

On December 20 I incised a subperiosteal collection of pus over the squamous portion of the temporal bone, and on December 27 the patient was admitted to the hospital for the fourth time. Examination of the eye, neurologic observation and laboratory tests gave normal findings. The Kobrak shock tests revealed a dead right labyrinth. Roentgen examination at this time showed a destructive suppurative process of the right petrous pyramid, extending through the border in the anterior and inferior regions, following mastoidectomy on the right side, and extensive osteomyelitis of the right temporal bone (fig. 2).

Third Operation and Postoperative Course.—The operation performed on Jan. 2, 1934, revealed osteomyelitic bone from 2 to 2½ inches (5 to 6.5 cm.) beyond the necrosis shown on the roentgenogram as described by Mosher. The bone was chalky white, was interspersed with necrotic black areas and had an odor

Fig. 2 (case 1).—Roentgenogram made on Dec. 27, 1933, showing extension into the right petrous pyramid.

resembling that of infections due to Bacillus coli or Bacillus pyocyaneus. All diseased bone was removed, including bone 1 inch (2.5 cm.) beyond the line of edema. Two extradural abscesses were located. The wound was left wide open and packed with iodoform gauze.

Cultures made at the time of this operation yielded Staphylococcus albus haemolyticus in pure culture. It is interesting to recall that the original bacterium was Str. haemolyticus. No tubercle bacilli were seen. Pathologic examination of the bone confirmed the roentgen and clinical findings.

Convalescence from this operation, though not alarming, was interesting. The patient still complained of occasional headache, and the edema and tenderness were still present over the occipital bone and right zygoma. The dura was clean and granulating, but collections of pus were still present about the edges of the wound. On February 6 the headache increased, and the pain in the upper molar teeth recurred. The dentist removed the upper cuspid and the first molar tooth,

which were found to be normal. On February 9 the patient complained of intense headache, and her temperature was ranging higher. As she was apparently losing ground and the roentgen examination indicated progression of the disease, another operation was attempted.

Fourth Operation and Postoperative Course.—Tribromethanol (avertin) in amylene hydrate being used for anesthesia, the diseased portions of the right side of the frontal and the occipital bone, all the squamous portion of the temporal bone and most of the parietal bone were removed. Before excision could be extended beyond the line of edema, the patient collapsed on the table and operation had to be discontinued. With the help of stimulants, artificial respiration and immediate blood transfusion, she was revived and returned to the ward, but in poor condition. The wound was packed and left open and the scalp rolled pancake fashion with the idea of unrolling it later to close the wound.

After this experience the next subperiosteal collections of pus were incised down to the bone as they appeared, the old principle of incision and drainage being relied on. The first incision was made in the latter part of February, over the parietal bone at the vertex, the second at the hair line on the left side of the frontal bone, the third over the squamous portion of the temporal bone where it articulates with the frontal bone and the fourth posteriorly over the occipital bone. After I had followed this plan on two occasions, my attention was called to the fact that O. J. Dixon had advocated a similar method of procedure in an article published in March 1934. Encouraged by his splendid results, I continued my surgical treatment along these lines. During this period, incision of the left drum head for suppurative otitis media complicating osteomyelitis of the left temporal bone was necessitated. A swelling over the left frontal sinus subsided after application of cold compresses wet with solution of magnesium sulfate. It is interesting to note that as the disease progressed to the left side pain developed about the left upper molar teeth.

Fifth Operation and Postoperative Course.—Sequestrectomy was performed on May 5. Roentgen examination on June 29 showed considerable increase in rarefaction of the remaining bones of the vault since the last examination (fig. 3). The patient was discharged from the hospital on August 31, fourteen months after her first admission. Thirteen months after the first roentgen evidence of osteomyelitis there were definite signs of regeneration of bone in the noncraniectomized area (fig. 4). Twenty months after discharge, there was a marked improvement in the appearance of the bones of the calvarium but still no signs of regeneration of bone over the large defect on the right side. Roentgen examination in January 1937, approximately two and a half years after the patient's discharge from the hospital, showed the first regeneration of bone along the edge of the cranial defect. A roentgenogram taken on November 11 showed further extension of this regeneration (fig. 5).

At the time of writing, December 1937, the patient's weight shows an increase from a low of 65 pounds (29.5 Kg.) to 98 pounds (44.5 Kg.), which is close to her maximum weight. I rarely see her; she has no complaints and is able to perform her household duties.

It is interesting to note that, despite involvement of both temporal bones, hearing of the left ear for voice and whisper is still normal and hearing for the watch decreased only to 15 inches (38 cm.). An audiogram taken in October 1936 showed a loss of hearing on the right of 60.8 per cent and on the left of 12.8 per cent with the opposite ear masked.

Fig. 3 (case 1).—Roentgenogram made on June 29, 1934, showing the typical moth-eaten roentgenologic appearance of osteomyelitis of the skull and its extension to all bones of the calvarium.

Fig. 4 (case 1).—Roentgenogram made in April 1936 showing the area of bone removed and definite signs of regeneration of bone in the noncraniectomized area but no signs of regeneration of bone about the large defect.

Behrens, who mentioned some of the work done on this patient in a recent publication, stated that "this case is likely to stir up controversies." It was my impression at the time that it illustrated well a radical failure and a conservative cure, and I determined that my next patient with osteomyelitis should be treated along semiradical lines.

CASE 2.—S. W., a white man aged 22, was admitted to the Episcopal Hospital in July 1936, complaining of pain and swelling about the right eye. These symptoms were of seven days' duration, and he appeared to be acutely ill. His temperature was 102 F. His personal and his family history were irrelevant. Physical examination yielded essentially negative results, except for edema and exquisite

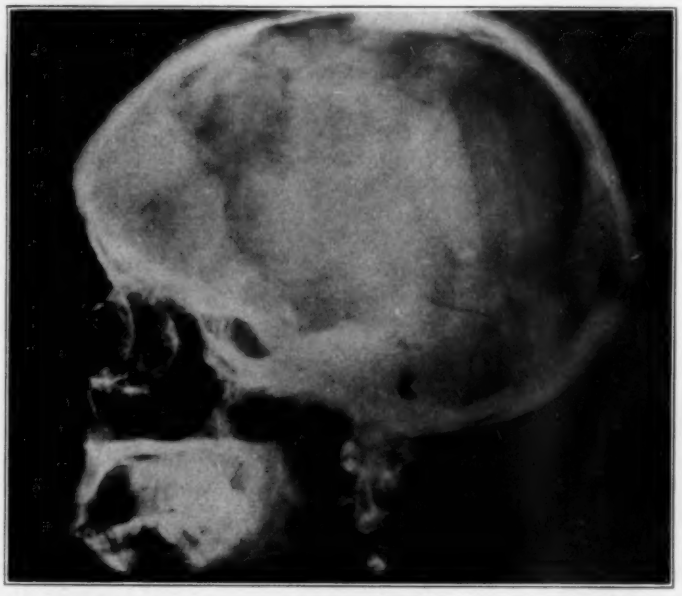

Fig. 5 (case 1).—Roentgenogram made on Nov. 11, 1937, showing increase in density and extension of new bone along the edge of the large cranial defect, especially in the frontal region. The layer of bone overlying the brain has increased in length and density.

tenderness over the right eye and eyebrow, extending upward for 1 inch (2.5 cm.). The floor of the right frontal sinus and the inner canthus were markedly tender. There was slight tenderness over the right maxilla, more marked over the canine fossa. Examination of the ears, pharynx and larynx revealed no positive findings.

Examination of the nose revealed a pronounced deflection of the septum to the right in contact with the middle turbinate. The middle turbinate bone impinged on the lateral nasal wall. The nasal mucosa was hyperemic and swollen, except over the tip of the right middle turbinate, where it was pale and edematous. A yellow, mucopurulent discharge was seen in the nares, on the floor of the nose and in the right middle nasal passage. The left frontal and maxillary sinuses were transilluminated clearly. The right maxillary sinus was dark and the

right frontal sinus questionable. Roentgen examination showed the left frontal sinus hazy, the right clear; the right ethmoid sinus cloudy, the left normal; the right maxillary sinus markedly cloudy, the left clear, and the sphenoid sinuses normal. Examination of the eye revealed no evidence of a choked disk. Neurologic examination indicated no gross pathologic changes. Laboratory findings, including a Wassermann test, were negative except for leukocytosis (14,950 cells). The diagnoses were deflected septum (obstructive) and acute suppurative multiple sinusitis involving the right frontal, ethmoid and maxillary sinuses.

First Operation and Postoperative Course.—On July 31 infraction of the right middle turbinate and intranasal antrotomy on the right were performed. Thick gray pus exuded from beneath the middle turbinate bone after infraction. Because of increased tenderness over the right eye, with swelling, pain and tenderness of the right eyelid, an incision of the upper eyelid and a right frontal sinuotomy were advised four days later but were refused by the family.

Second Operation and Postoperative Course.—Finally, after much persuasion by those in charge and insistence by the patient, a second operation, incision of the orbital abscess and frontal sinuotomy on the right was done on August 7. A 2 cm. opening was made in the anterior wall above the maxillofrontal suture. The bone was markedly hyperemic, a condition which suggested an early stage of osteomyelitis. The sinus was cleansed of pus and a rubber drain inserted in it and in the orbital abscess. Methenamine, 20 grains (1.29 Gm.) four times a day, was prescribed. Cultures of pus from the sinus and the orbital abscess showed Staphylococcus aureus haemolyticus. Cultures of the blood were negative. Between August 8 and 14 the patient's condition improved markedly, and after a lavage of the antrum on August 12 he was allowed to be out of bed. His average temperature was 99.4 F. On August 16 the patient suffered a generalized convulsion lasting one hour, after which he complained of headache and became markedly irritable. Examination of the spinal fluid showed 51 cells per cubic millimeter and increased tension. On August 19 a neurosurgical consultant reported that there were no localizing neurologic signs and that the probable cause of the convulsion was widespread extracranial hyperemia with edema, aggravated or caused by the extreme heat and the ingestion of large amounts of fluids.

On August 20, examination of the eye revealed some engorgement of the veins and some haziness of the margin of the disk on the temporal side in the right eye. Between this date and August 24 irritability and headache diminished. The patient was nauseated and vomited on two occasions. Another neurosurgical consultation on August 26 revealed no localizing signs of a pathologic condition in the brain, and it was the opinion of the consultant that the patient showed improvement mentally and physically. By September 2 his general and local condition had improved so markedly that he was discharged from the hospital, only to be readmitted on September 5 because of recurrence of the severe frontal headaches and vomiting. These symptoms, together with nausea, irritability and an apparently voluntary tremor of the hands, persisted for about twenty-four hours.

Third Operation and Postoperative Course.—A submucous resection was performed on September 9, and thick yellow pus was observed draining from the frontonasal duct. Recovery was uneventful, and the patient was discharged on September 12. The external opening in the frontal sinus was still suppurating. As frontal headache, nausea and vomiting recurred on September 21, he was readmitted on September 25. Roentgen examination on September 25, forty-nine days after the sinuotomy, revealed a dark left frontal sinus and osteomyelitis extending into the frontal bone above the right frontal sinus, involving both tables.

Fourth Operation and Postoperative Course.—On September 26 a radical bilateral operation on the frontal sinuses (Skillern) was performed. The anterior and posterior walls of both sinuses were found to be the seat of an osteomyelitic process with necrosis involving portions of both walls. The anterior walls and necrotic areas, with 1 inch (2.5 cm.) of surrounding bone from the posterior walls, were removed. In accordance with the suggestion of McKenzie for bone drainage, two ¾ inch (2 cm.) openings were made with the trephine in the frontal bone 2½ inches (6.5 cm.) beyond the necrosed areas.

Between September 26 and October 30 the general and the local condition improved. The patient was allowed to be out of bed for a limited time. His temperature ranged from 97 to 99.1 F. Nausea and vomiting recurred on November 1. He was irritable and failed to cooperate with the resident physician and the nurses. Roentgen examination on November 3 showed further involvement of the inner table of the frontal bone and a possible sequestrum in the rarefied area.

Fifth Operation and Postoperative Course.—On November 5 the necrotic bone and small sequestrums were removed. The possible sequestrum in the rarefied area, suggested by the roentgenogram, could not be identified. After this operation the patient was totally irrational and even maniacal at times. He shouted at his parents and had to be restrained. His general physical condition was fair. Examination showed more engorgement of the veins in the right eye than usual. In the left eye the edges of the disk were not clearly outlined. The disk was not choked in either eye.

On November 10 the patient was again seen by the neurosurgeon but on this day appeared rational and cooperative. The consultant did not believe that abscess of the brain was present at this time and advised looser dressings to avoid pressure on the occipital artery. On November 24 a neurologist reported definite evidence of multiple abscess of the brain.

Sixth Operation and Postoperative Course.—On November 27 exploratory craniotomy was performed, with removal of more necrotic frontal bone and evacuation of a deep, densely encapsulated abscess of the left frontal lobe. At this time the neurosurgeon believed that the date of invasion of the cranial contents coincided with the beginning of infection in the frontal sinus. The patient died forty-eight hours after operation. No autopsy was permitted.

LOCAL NONSURGICAL MEASURES

Many measures were tried, the first being continuous irrigation with diluted solution of sodium hypochlorite. This was discontinued as having no apparent effect on the progress of the disease. The lack of effect agrees with Wilensky's observations in 1922 that successful results are not to be expected from this treatment because the structure of the bone prevents a successful contact between the solution and the infected area, whether the latter is in the cells of the cancellated bone or in the thrombi. Bacteriophage, used locally for both patients and subcutaneously and by mouth for the patient with the sinal complications, had no effect on the progression of the disease and seemed to irritate the dura.

M. A. Stewart has suggested that the bacterial exotoxin in cases of staphylococcic infection may be of protein nature and that, if so, a 0.25

per cent aqueous solution of trinitrophenol will render it insoluble and consequently inert. This solution, combined with 8 per cent glycerin to reduce its surface tension, was used as a wet dressing in both cases and certainly resulted in smoother, cleaner granulations, with the glistening sheen observed in maggot-treated infections. Its use was continued to the termination of the infectious process and did not irritate the dura. As an adjunct to this treatment, Stewart suggested spraying an aqueous suspension of calcium carbonate into the wound to stimulate phagocytosis. This procedure also was tried, but it was discontinued as being impractical in the case of a lesion of the skull. During convalescence infra-red irradiation was used, as advised by Furstenberg, to stimulate regeneration of the bone.

GENERAL MEASURES

Autogenous staphylococcus vaccine was used in the first case, but its use was discontinued because it had no apparent effect on the progression of the disease. Staphylococcus toxoid was used in the second case, with disappointing results, which coincided with the recent experience of Buckman and with that of Debré, Bonnet and Thièffry. Staphylococcus antitoxin, as advocated by Dolman, is indicated in cases of fulminating infection to increase resistance rapidly. Attempts at immunization should be directed toward the rapid establishment of resistance to invasion by the organism rather than to the slow production of an immunity depending solely on antitoxins. Repeated blood transfusions, as suggested by L. J. Martin, were certainly helpful in both cases. In addition to the measures mentioned, general ultraviolet irradiation and administration of cod liver oil to stimulate calcium metabolism, iron and ammonium citrates, as suggested by Furstenberg, and stimulant drugs were used with benefit.

COMMENT

Case 1.—Case 1 is illustrative of the usual progressive, or spreading, type of osteomyelitis of otitic origin. The condition followed operation for an acute exacerbation of a chronic process. The original infection in the mastoid, due to a streptococcus, was followed by infection with Staph. albus haemolyticus when the osteomyelitis became progressive. The latter organism was probably of low virulence; hence, by the time the lesion on the right side had been extensively removed, the body had had a chance to raise its immune bodies to a level sufficient to combat the infection and to prevent its further intracranial spread. As there were two extradural abscesses on the right side, the possibility of this complication on the left side, which was similarly involved, was to be expected. That it was not involved was, in my opinion, due

to the fact of increased resistance. According to Schilling, abscess of the scalp in osteomyelitis always has an extradural abscess beneath it.

The importance of general measures and increased resistance was dramatically brought to my attention when after two months of discouraging radical treatment and four months of conservative treatment, my patient suddenly, in a few days' time, became brighter, was relieved of persistent headache and regained her appetite and at the same time the discharge from the wound became serous rather than purulent.

Regeneration of bone occurred first on the side on which no operation had been performed and did not occur on the craniectomized side for two and one-half years. Furstenberg stated that new bone can be formed by either the dura or the periosteum if bone dust is present, which accounts for regeneration beginning in the center of the defect in areas from which the skull has been removed. In my patient regeneration proceeded from the edges, despite two operations in which bone dust was deposited and daily infra-red irradiation for three years. Some factor was evidently missing in this case.

As I have stated, in order to afford better drainage, the scalp, which had been rolled pancake fashion, was not replaced over the dura. The patient later refused plastic operation.

In my opinion, not only is it necessary for bone dust or particles to come in contact with the dura, but they must be fixed there by the overlying periosteum of the scalp.

Case 2.—Case 2 was a typical case of spreading, or progressive, osteomyelitis of the skull complicating acute disease of the frontal sinuses. The roentgen evidence and history prove that the condition cannot be attributed to too early surgical intervention. Owing to delay in obtaining consent of the parents, the operation was late. The removal of the foci in the sinuses and the necrotic bone and the distant trephining, as suggested by Dan McKenzie, evidently arrested the osteomyelitic process but did not prevent infection of the cranial contents. The current of infection is more inward than outward. In contradistinction to the view expressed by the neurosurgeon, I believe that the date of deep invasion of the cranial vault coincided not with the time of the sinus infection but with the convulsion, which occurred twenty-three days after the onset of the sinus infection, and thus constituted a complication of the osteomyelitis. The sinal infection was on the right; the osteomyelitis was bilateral, and the abscess of the brain was on the left. It is my impression that the osteomyelitis spread more rapidly after the operation in which an opening was made in the anterior wall of the right frontal sinus.

In the literature, Ross Hall Skillern advised an opening in the anterior wall and Hastings in the floor of the frontal sinus. Lynch was the first to emphasize the importance of leaving the periosteum on the

anterior wall of the frontal sinus undisturbed. The anterior wall is frequently diploic, and the inferior is made up of thin, compact bone.

From my experience with this case I am of the opinion that drainage through the inferior wall would not have involved as much injury to the periosteal blood supply and as much invasion of diploic bone as was caused by drainage through the anterior wall. In this connection it must be kept in mind that osteomyelitis involves all bony walls of a sinus. In order to obliterate definitely the frontal sinus as a focus of infection in cases of this type I believe that the posterior wall should be removed with the anterior wall. Extensions into the cranial vault occur through the posterior wall.

CONCLUSIONS

True localized osteomyelitis may be more common than has been hitherto suspected. This fact should be borne in mind and is of importance when one advocates a simple mastoidectomy or rasping of the frontonasal duct.

I believe the best surgical procedure in the treatment of acute exacerbations of chronic processes involving the mastoid is a complete simple mastoidectomy, or the so-called modified radical operation, followed after subsidence of acute symptoms by surgical treatment of the middle ear and plastic work. The danger of spreading osteomyelitis may thus be added to Kopetzky's reasons for advocating the two stage operation. In cases of acute frontal involvement the surgical treatment indicated, if any is necessary after intranasal procedures, is sinuotomy of the inferior wall. If operation on the sinus is intended to remove a focus of infection of osteomyelitis, both the anterior and the posterior wall must be removed to prevent extension of the infection into the cranial vault.

Osteomyelitis of the skull has been divided by various writers into two or three types. It is more important to recognize the possibilities of diffusion than to spend time and effort in attempting a differentiation of types.

A new symptom of possible spreading osteomyelitis of the temporal bone is described, namely, the occurrence of neuralgia about the upper molar teeth of the affected side, probably a reflex sensation, due to irritation of the meningeal branch of the fifth nerve. Facial paralysis due to a swelling of the fallopian canal and pressure on the nerve can be a sign of localized osteomyelitis.

Once diagnosis has been established, radical operation with wide resection of bone should be done for a cure.

One may expect any osteomyelitic lesion in the skull to cover pachymeningitis or to be complicated by an abscess of the brain. The first

surgical step in the treatment of these two frequent complications of osteomyelitis is removal of the foci of infection. Radical removal of bone does this, and, in addition, if abscess of the brain is present, the resulting decompression will permit a better localization of the abscess and likewise prevent compression of the brain and its vital centers.

For the regeneration of bone following craniectomy, it is necessary not only that bone dust or particles should come in contact with the dura but that they should be fixed there by the overlying periosteum of the scalp.

The importance of general supportive treatment and measures to increase resistance cannot be overestimated.

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AN UNUSUAL FORM OF NASAL HEADACHE

W. G. MUSSUN, M.D.
CLEVELAND

I have observed a type of headache (apparently neuralgic in character) which is concomitant with an inflamed state of the mucous membrane of the anterior third or half of the middle turbinate of the nose. This condition is not rare in my experience, and yet I have been unable to find any literature that describes the symptoms and associated pathologic conditions that have been present in the cases passing under my observation. In the last five years I have seen from 20 to 25 patients with the symptom complex which I shall endeavor to describe.

These victims complained of pain at the nasal side of the orbit, which not infrequently involved the frontal region on the side of the head in which the pathologic condition existed. This is the rule, but I have 1 patient who experiences a like pain on the side of the head opposite the lesion. In some patients the pain radiates from the frontal to the parietal and occipital regions, and it sometimes extends to the neck and spreads over the upper third of the back of the corresponding side. It is usually unilateral, rarely bilateral.

There seems to be no definite time in the twenty-four hour period at which suffering begins, except that some patients are awakened by it or it is present when they awake. The pain usually lasts for several hours, but in a small number of persons it persists for a much longer time—even for weeks or months—with occasional exacerbations that torment the sufferer for hours before subsiding. Most of the histories indicate that the pain appears intermittently but with no definite regularity. I was unable, moreover, to obtain case histories suggesting a definite relationship with weather conditions, seasons, possible allergic states, occupation or any bodily functions except sleep, as already suggested. No doubt some factor which causes local congestion is the primary cause of these headaches. Physical activity seems to increase their severity.

The patients who came under my observation were adults, most of them well past middle life. A small percentage gave a vague history of pains in the head since childhood. Most of them noticed that the pain began mildly and gradually became more severe as they advanced in years. They experienced no other symptoms except slight photophobia and lacrimation and the general malaise that one expects to find in any condition attended by severe pain.

The pathologic condition appears to be a marked inflammation of the mucous membrane of the anterior third or half of the middle turbinate. The membrane is apparently dry, and no discharge of any kind accompanies the inflammation. There is no visible evidence of abnormality of the adjacent tissues. I have seen and examined these patients between the attacks and the mucous membrane of the involved turbinate is a normal pink and blends insensibly into the mucous membrane in the corresponding nasal cavity. I am unable to offer any explanation as to why the inflammation is limited to the middle turbinate or why it does not spread and involve more tissue in this region.

The diagnosis is decided by history of pain in the region described, inflammation of the turbinate, slight photophobia, general malaise and lack of evidence of any other condition which might cause the attack. The question of differential diagnosis arises. This type of headache might be confused with the so-called vacuum sinus headache or neuralgia of the anterior ethmoid (nasociliary), the supraorbital or the sphenopalatine nerve. I think the question of sinusitis other than vacuum sinusitis can be dismissed, since there is absence of the usual clinical findings, except pain. In frontal vacuum sinusitis the pain is in the frontal region, present on arising and made worse by the use of the eyes. There is definite tenderness near the inner angle of the orbit (at the point of attachment of the superior oblique muscle). In vacuum sinusitis of the anterior ethmoid sinuses, the pain is usually felt between or behind the eyes, and there is definite tenderness at the lacrimal bone. The patient, however, complains of feeling as though sand were in the eyes.

Patients with nasociliary neuralgia complain of pain between the eyebrows, which extends somewhat above the superciliary ridge and down along the nasal bone, and sometimes there is pain in the orbit and the nasal fossa. As a rule, it is not intense. It occurs irregularly and may be associated with coryza. Sluder stated that the anterior ethmoid branch of the nasociliary nerve is superficial after entering the nasal cavity. At this site there may be hyperplasia, giving rise to inflammation or nerve pressure, which may cause pain. It is the external branch of the anterior ethmoid nerve which is a branch of the nasociliary nerve and supplies the anterior end of the middle and the inferior turbinate.

Pain associated with involvement of the sphenopalatine ganglion is felt in the posterior portion of the nose and involves the adjacent region and the teeth. Supraorbital neuralgia also offers little difficulty in diagnosis, because of its location and the tenderness of the nerve when palpated.

TREATMENT

Anesthetizing the inflamed middle turbinate with cocaine gives relief, which may, and has in some instances, lasted for a long period. If the relief is slight and there are recurrent attacks, removal of the adjacent structure (usually the septum) which may cause pressure on the turbinate usually gives the desired result. If this procedure proves to be ineffectual, it will be necessary to anesthetize the inflamed turbinate a few times after the postoperative inflammation has subsided and then, if pain persists, amputate the anterior end or inflamed portion of the turbinate, a procedure which in my experience has resulted in permanent relief.

816 Rose Building.

THROMBOSIS OF THE LATERAL SINUS

A STUDY OF FIFTEEN CASES, INCLUDING HISTOLOGIC EXAMINATION

JOSEPH G. DRUSS, M.D.
NEW YORK

When writing on any subject in medicine, one who advocates therapeutic measures has a natural tendency to report the good results obtained therefrom and a reluctance to report the poor results. To a great extent this applies to the discussion of sinus thrombosis, the subject with which this communication is concerned. The literature is replete with reports of cases, varying in number, in which the outstanding feature is a discussion of the cures effected. Relatively few articles, however, deal with the pathologic picture observed post mortem. In my opinion many of the questions with regard to the treatment of sinus thrombosis will be settled by a clearer understanding of the basic pathologic picture of this disease.

Within the eight year period from 1928 to 1935 inclusive, 62 patients with sinus thrombosis were treated by the otologic service of the Mount Sinai Hospital. Of these, 44, or 71 per cent, recovered and 18, or 29 per cent, died. From the 18 who died, 15 temporal bones (one from a patient) were procured and sectioned serially. An analytic study of the 15 cases in which these specimens were taken constitutes the basis of this publication.

The age at which the disease occurred varied from 2 months to 55 years. Eight, or more than half of the patients, were within the first decade. There were 7 males and 8 females. The left ear was involved in 10 patients and the right ear in 5. Reports in the literature, however, show a greater incidence in the right ear than in the left.

OTITIS MEDIA AND ASSOCIATED INTRACRANIAL COMPLICATIONS

The illness was ushered in by acute otitis media in 10 instances and by chronic otitis with an acute exacerbation in 5.

Acute Suppuration of the Middle Ear.—The onset of acute aural symptoms occurred from seven days to eight weeks prior to the patient's admission to the hospital. In 1 instance otitis media occurred two days after admission. (The patient had been admitted for a condition requiring medical treatment.) As is well known, signs and symptoms

From the Otologic Service and the Laboratories of the Mount Sinai Hospital.

of otitic sepsis most often make themselves manifest from ten to eighteen days after the onset of acute otitis. Occasionally, however, evidence of sepsis, particularly when secondary to primary thrombosis of the jugular bulb, appears within a few days after the onset of the otitis. In one of the cases a shaking chill, with a temperature of 103 F., occurred one day after the onset of the otalgia. Furthermore, in primary thrombosis of the bulb the aural symptoms may on rare occasions be so scant and evanescent that a diagnosis is made with difficulty. In 1 of the cases, for example, there was at no time discharge from the middle ear in spite of myringotomy. Also, in a comprehensive report by Maybaum and Goldman [1] of 20 cases of primary thrombosis of the bulb 1 case is cited in which no aural discharge was present at any time. Thus, while a discharge from the middle ear is most common, its presence is not prerequisite for the establishment of the diagnosis of otitic sepsis.

Chronic Suppuration of the Middle Ear.—A chronic discharge from the ear, the duration ranging from 1 to 36 years, was observed in 5 instances. In each instance, however, the sinus thrombosis was precipitated by an acute exacerbation. According to most authorities (Haymann [2]) sinus thrombosis is more prevalent after acute than after chronic suppuration of the middle ear. They are of the opinion also that the incidence of recovery is greater after acute than after chronic suppuration.

Associated Intracranial Complications Present on Admission.—In 9 of the 15 cases signs of intracranial complications other than those due to sinus thrombosis were manifested on admission. In 6 cases there was evidence of frank meningitis, with organisms demonstrated on smear or culture, or evidence of meningeal irritation, such as rigid neck and Kernig's sign. In 2 of these 6 cases there were, in addition, signs of abscess of the brain, the diagnosis being established subsequently by operation. A dead labyrinth was present in 1 case and cavernous sinus thrombosis in another. One patient had been operated on for sinus thrombosis in another institution. Other grave complications existing on admission were acute endocarditis, in 1 instance, and uremia with suppression of urine, in another. Both these conditions ceased without operation a few days after admission. The mortality from sinus thrombosis of 29 per cent, which has been mentioned, would

1. Maybaum, J. L., and Goldman, J. L.: Primary Jugular Bulb Thrombosis: A Study of Twenty Cases, Arch. Otolaryng. **17**:70 (Jan.) 1933.

2. Haymann, L.: The Otogenic Sinus Thrombosis and Otogenic General Infection, in Denker, A., and Kahler, O.: Handbuch der Hals-, Nasen- und Ohrenheilkunde, Berlin, Julius Springer, 1925, vol. 3, p. 55.

naturally have been considerably lowered if in the compilation the cases in which the more serious complications were present had been omitted.

OPERATION

Findings at Operation on the Mastoid.—A simple mastoidectomy was performed in 11 of the 15 cases and a radical mastoidectomy in 1. Operation was performed either at some time prior to the exploration of the sinus, for an existing mastoiditis, or as a preliminary step at the time of the exploration. Marked sclerosis of the mastoid was encountered in 2 instances and an acellular undeveloped mastoid in 1 (age of patient, 6). A forward lying sinus was noted on 2 occasions. Attention is given to these anatomic conditions because they are frequently considered etiologic factors in the production of sinus thrombosis.

The gross pathologic conditions in the mastoid observed at operation varied from slight, if any, inflammation to marked necrosis of the bone. One would naturally expect these findings to be of a bizarre and variable nature, depending among other factors on the severity of the infection. A perisinal abscess with or without gross evidence of disease in the sinus plate was observed in 5 cases. In 1 instance the plate appeared normal, though after its removal the underlying venous wall was found to be definitely diseased. The appearance of this plate, as is well known, is not always a guide to the nature of the pathologic condition of the adjacent vein.

On 2 occasions the sinus was exposed during the mastoid operation; on 1 occasion it was exposed as an aid in diagnosis to determine the condition of the venous wall, and on the other it was exposed inadvertently. In both instances the wall showed no abnormality. However, in each case about one week later, at the time of operation on the sigmoid sinus, thrombophlebitis at the site of exposure was encountered. The question naturally arises whether exposure of a normal sinus to an infectious process predisposes it to phlebitis and thrombosis. Brunner [3] expressed the belief that the incidence of sinus thrombosis is not enhanced by such exposure. Moreover, it has been demonstrated by animal experimentation [2] that factors other than mere exposure are essential for the formation of thrombosis.

Operative Procedure for Sinus Thrombosis.—In the surgical treatment for sinus thrombosis the jugular vein was ligated as a first step in the operation only in those cases in which the diagnosis had been fully established. In those instances in which the diagnosis was still doubtful or in which there was bilateral suppuration of the middle ear

3. Brunner, H.: Postoperative Sinus Thrombosis, Arch. Otolaryng. **10**:217 (Sept.) 1929.

and the question as to the side of involvement was not fully settled, the sigmoid sinus was first investigated. If the sigmoid sinus was found to be diseased, the jugular vein on that side was ligated first and the operation on the sinus then completed. The latter procedure consists in tamponing the wall of the vessel at points beyond the apparent disease, one in the region of the torcular Herophili and the other in the region of the jugular bulb. The vein was next incised, the thrombus removed and as much of the outer wall of the vessel resected as possible. Each tampon was then removed individually in order to determine, and to reach if necessary, the limits of the apparent thrombosis or phlebitis. The tampon was reinserted when free bleeding from the vein was obtained. It must be stated that, since an obturating thrombus was not encountered in every case, the foregoing steps in this operative procedure could not always be carried out rigidly.

Because of continued sepsis, secondary operation on the sigmoid sinus was performed in 3 cases, each time within four to six days after the original operation on the sinus. In this procedure, as in the first, an attempt was made to eradicate the focus in the sinus which was believed to be responsible for the continued sepsis. This focus, however, could not always be demonstrated. Of the 3 cases in which secondary operation was required, thrombosis at the end toward the torcular Herophili was encountered in 1 and free bleeding at this site in 2. In none was there bleeding from the jugular bulb. On the contrary, by applying pressure over the jugular vein in each instance, purulent exudate could be milked through the jugular bulb into the mastoid wound.

Findings at Operation on the Sigmoid Sinus.—Operation on the sigmoid sinus was performed in 8 cases. The sinus was diseased in 7 of these. It was described as being discolored, dull, lusterless, thickened, necrotic or covered with granulations. The nature and extent of disease in the vein differed in each case. An obturating thrombus was encountered in 3 cases, purulent thrombosis in 2 and free bleeding from the sinus in 2. In 1 instance the wall of the sigmoid sinus throughout its extent showed no gross evidence of disease, and free bleeding was induced when it was incised; yet the corresponding jugular vein on exposure was found to be grayish white, collapsed and bloodless. In this case the jugular bulb was in all probability the primary site of infection, extension from this structure to the jugular vein, rather than to the sigmoid sinus, having taken place secondarily. In 5 cases the jugular bulb was found thrombosed. In none of these was bleeding encountered from the bulb even after attempts at removal of the thrombus by curettement.

SUMMARY OF THE CHIEF SYMPTOMS

The temperature curve varied. In some cases it showed the characteristic fluctuations, ranging from 97 to 106 F.; in others it was sustained at a high level, and in still others it was irregular. In 6 cases there was evidence of pathologic changes in the fundi, ranging from indistinctness of the margins of the head of the nerve to measurable papilledema. Chills were present in 5 cases and enlarged cervical glands in 7. A blood culture was made in 10 cases, in each of which it was positive. The hemolytic streptococcus was recovered in 8 and the type III pneumococcus in 2. These two organisms, as is well known, are the most frequent invaders of the middle ear. The value of blood culture as an aid in diagnosis cannot be overemphasized. Metastases were present in only 2 cases and were multiple in both. It is thus suggested that the absence of metastases is not necessarily an indication of a good prognosis.

The duration of illness after the original operation on the sinus varied from seventeen hours to two and one half months, the average being seventeen days.

ASSOCIATED COMPLICATIONS REVEALED BY POSTMORTEM EXAMINATION

The following intracranial complications occurred either individually or in combination: meningitis in 12 cases, petrositis in 7, labyrinthitis in 4, abscess of the brain in 2, subdural abscess in 1 and cavernous sinus thrombosis in 1. Of significance is the observation of acute endocarditis on 3 occasions.

Histologic Changes in the Temporal Bone.—Naturally the character of the histologic changes in the temporal bone varied, depending among other factors on the duration and severity of the illness and on the reparative processes of the infection. This is particularly true for the tympanum and the mastoid, where the severity of the lesion ranged from relatively mild inflammation of the mucosa to extensive necrosis. The inflammatory process in the tympanomastoid area was acute in practically every case. In 5, however, there was evidence of previous chronic infection. Thus cholesteatoma was observed in 4 instances. While the cholesteatomatous matrix was not in close contact with the sigmoid sinus or jugular bulb in any of the cases, the inflammatory reaction secondary to the cholesteatoma had nevertheless extended to these structures in practically every case. There can be no question that the cholesteatoma when present had a direct bearing on the causation and course of the thrombophlebitis.

Obviously, since operations had been performed on the mastoid and on the sigmoid sinus in most of the cases, the character of the pathologic

picture which these structures subsequently presented was necessarily altered by these procedures. For the same reason the manner in which the infection reached the sigmoid sinus could not always be ascertained histologically. An unusual opportunity to study these pathologic phases in their unaltered state, however, was afforded in 3 cases (cases 9, 11 and 15), in which no operative procedures on either the mastoid or the sigmoid sinus had been performed. In these 3 instances it was observed that the infectious process did not extend through the mastoid to involve the sigmoid sinus. The latter was relatively free of disease, the jugular

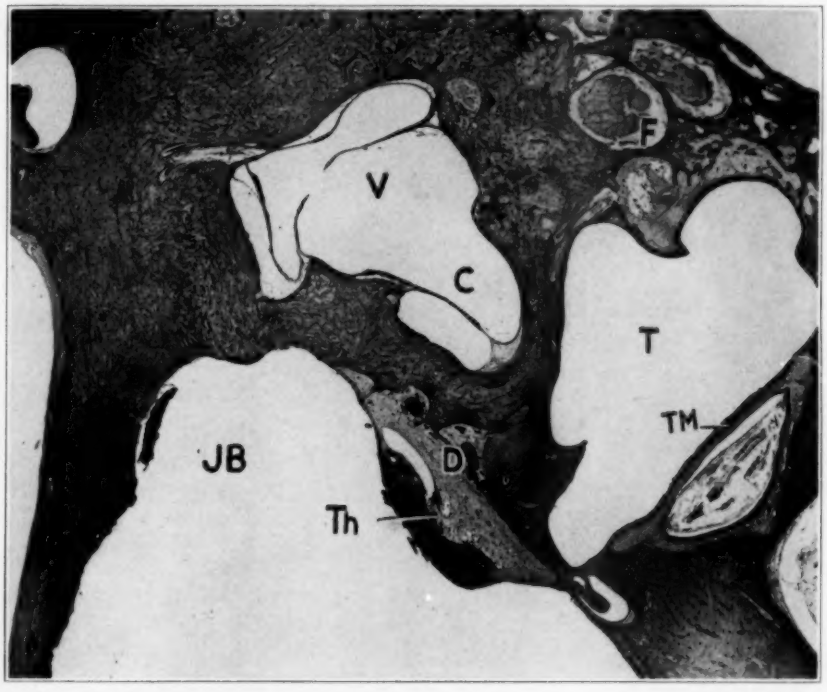


Fig. 1.—Localized thrombophlebitis in the jugular bulb. The jugular dome is high and exceedingly thin in the region of the floor of the tympanum. Multiple dehiscences can be seen. *C* represents the cochlea, *D* the dehiscences, *F* the facial nerve, *JB* the jugular bulb, *T* the tympanic cavity, *TM* the tympanic membrane *Th* the thrombophlebitis and *V* the vestibule.

bulb being the site of involvement. In 2 of these the infection extended from the hypotympanum to the jugular bulb by way of dehiscences (case 15, fig. 1) or communicating blood vessels (case 11) in the jugular dome. In the third the bulb was reached by way of retrograde extension in the petrosal sinuses secondary to cavernous sinus thrombosis (case 9).

A not infrequent method by which the infectious process in the tympanum advanced to the jugular bulb, disclosed by the study of the remaining cases, was direct invasion, with necrosis of the jugular dome (fig. 2). With this method of propagation the inflammatory process in the form of granulations and fibrous tissue was often continuous from the hypotympanum to the wall of the vein in the bulb. It should be borne in mind that extension of the infection may take place directly from the eustachian tube to the jugular bulb, the routes here being quite similar to those which lead from the tympanum (case 1, fig. 3).

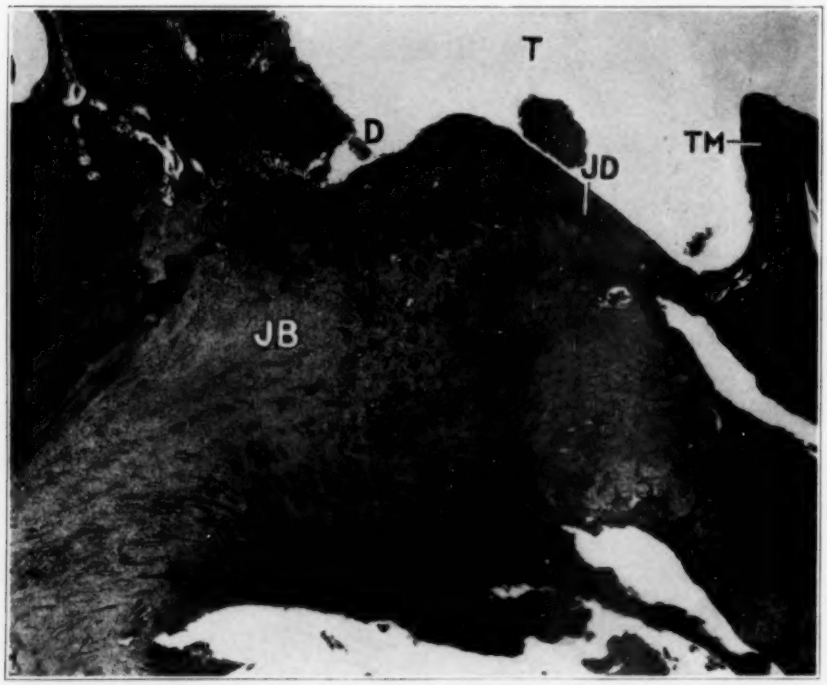


Fig. 2.—Vertical section through the tympanum and jugular bulb, demonstrating thrombophlebitis in the jugular bulb. There is a large defect in the posterior half of the jugular dome at *D*. The inflammatory process is a continuous one from the hypotympanum to the venous wall. *JB* represents the jugular bulb, with a thrombus; *T* the tympanum, with inflammatory exudate within; *TM* the tympanic membrane, and *JD* the jugular dome.

The inflammatory picture observed in the sigmoid sinus varied in character from that of a localized area of thrombophlebitis, with mild thickening and cellular infiltration of the wall of the vessels, to one extending from the bulb to beyond the torcular Herophili, with marked necrosis of all the layers. In the cases in which operation had been performed the inflammation usually extended along the wall of the vessel

Fig. 3.—Purulent thrombophlebitis in the bulb, with necrosis of the venous wall. There is little or no evidence of organization in the thrombus. *C* represents the cochlea, *ET* the eustachian tube, *F* the facial nerve and *T* the thrombus within the jugular bulb.

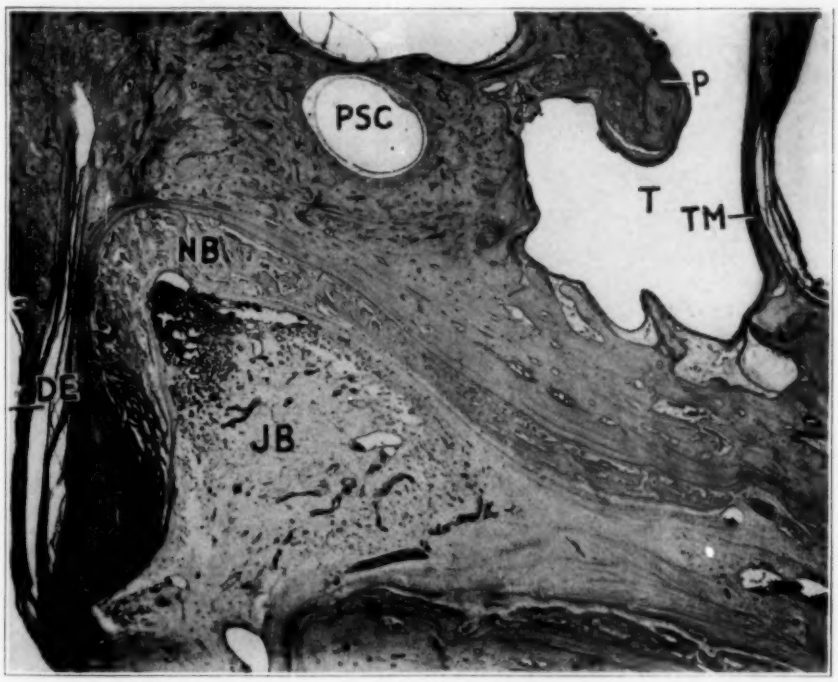


Fig. 4.—Healing stages of a thrombophlebitic process in the jugular bulb. Note the formation of new bone along the margin of the jugular bulb, encroaching on its lumen. *DE* represents the ductus endolymphaticus, *JB* the jugular bulb, *NB* the new bone formation, *P* the promontory, *PSC* the posterior semicircular canal, *T* the tympanic cavity and *TM* the tympanic membrane.

for a considerable distance beyond the site of operation (site of incision or resection of the wall). The jugular bulb was involved in practically every case of the series. Here, also, the degree of inflammation ranged from mild phlebitis to extensive ulceration of the wall of the vessel (figs. 2 and 3), and the thrombus varied from a small isolated plaque (fig. 1) to one occupying the entire bulb (fig. 4).

Thrombophlebitis of the superior and inferior petrosal sinus was not an infrequent occurrence (figs. 5 and 6). It was observed 7 times in each of these veins. The thrombophlebitis was either secondary to an extension from the sigmoid sinus or secondary to direct invasion by a

Fig. 5.—Thrombophlebitis of the superior petrosal sinus. The inflammatory process in the petrous pyramid can be seen communicating directly with the sinus by means of a fibrous tract in the bone. *FT* represents the fibrous tract in the bone and *Th* the thrombus in the superior petrosal sinus.

suppurative process in the petrous pyramid. Of especial interest is the possible spread of infection from the petrosal sinuses to the sixth nerve. Thus, in 1 of the cases (case 7) in which there was palsy of the external rectus muscle, thrombophlebitis of the superior petrosal sinus with extension of the inflammation to the sixth nerve as it coursed through Dorello's canal was observed histologically.

Although the emissary vein was not available for study in every case and the frequency of its involvement in the series could not therefore be

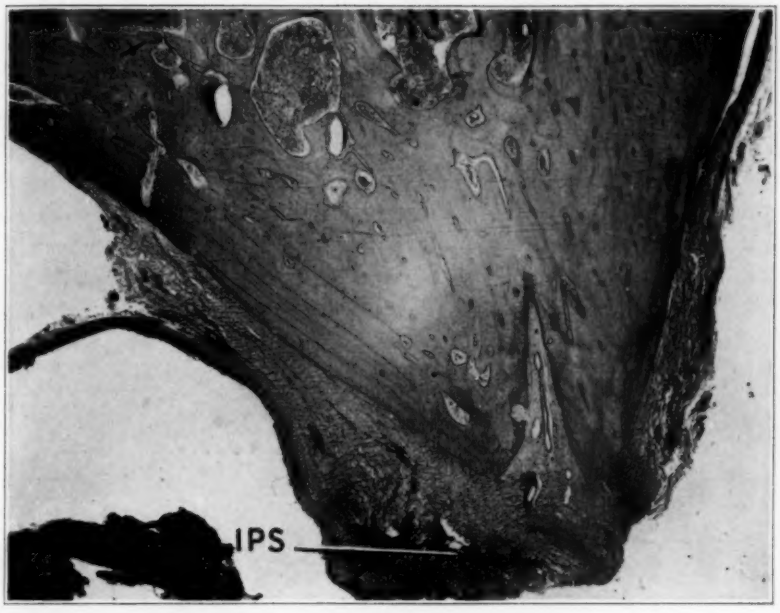


Fig. 6.—Thrombophlebitis of the inferior petrosal sinus. Note the communicating venous tributary, partially thrombosed, in the adjacent bone. *IPS* represents the inferior petrosal sinus.

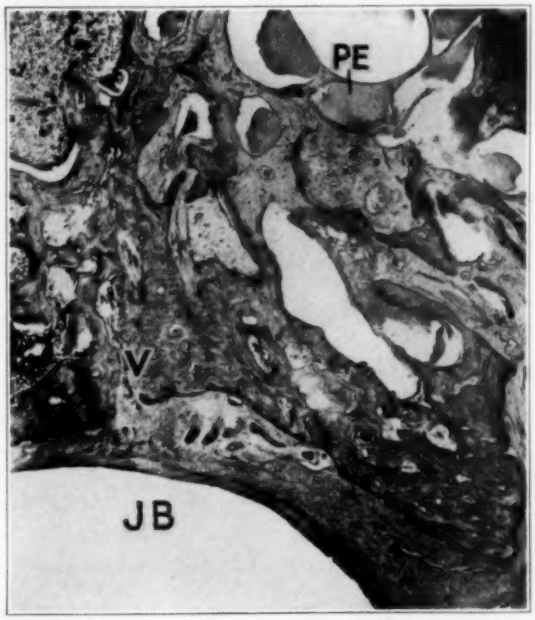


Fig. 7.—Thrombophlebitis of a venule in the bone which empties into the jugular bulb. *JB* represents the jugular bulb, *PE* the purulent exudate in the pneumatic spaces of the petrous pyramid and *V* the venule (thrombosis).

determined, it was nevertheless seen to be thrombosed in 3 instances. This vein is probably more frequently affected than is commonly believed (Ziegelman [4]). Similarly, other smaller tributaries of the sigmoid sinus and of the jugular bulb (figs. 6 and 7) which traverse the temporal bone also were thrombosed, thrombosis having taken place in the majority of instances by retrograde extension. It is worth noting that the small nutrient arteries within the inflamed venous wall often exhibited endarteritic changes. As a result of this phenomenon the blood supply to the venous wall is impeded, and necrosis of the latter, with disintegration of the adjacent thrombus, when present, is thereby favored.

Fig. 8.—Section demonstrating a large peribulbar abscess with necrosis in the adjacent petrous pyramid. *CA* represents the carotid artery and *PA* the peribulbar abscess.

The suppurative process in the tympanum and the mastoid extended into the petrous pyramid in 7 instances. In 4 of these the major expression of the disease in the pyramid was manifest in the sublabyrinthine or peribulbar region, a peribulbar abscess being present in each (fig. 8). Owing to the close proximity of the ninth and the tenth nerve to the jugular bulb, involvement of these nerves together with their respective ganglions was not unusual. The peribulbar inflammation occasionally extended to and included the labyrinth, the carotid canal, the adjacent soft parts of the neck and the base of the skull.

4. Ziegelman, E. F.: Surgical Importance of the Mastoid Vein in Infected Lateral Sinus Thrombosis, Arch. Otolaryng. **18**:298 (Sept.) 1933.

Acute suppurative labyrinthitis was found 4 times (fig. 9), the infection in the tympanum having reached the labyrinth through the oval and the round window or by way of erosion of the bony capsule of the semicircular canal secondary to an intermediary suppuration of the petrous pyramid. A definite causal relationship between the labyrinthitis and the sinus thrombosis was not established in any of the cases, the two conditions in all probability being independent of each other.[5]

Evidence of meningeal reaction—extensive inflammatory changes within and around the dura and purulent exudate in the internal

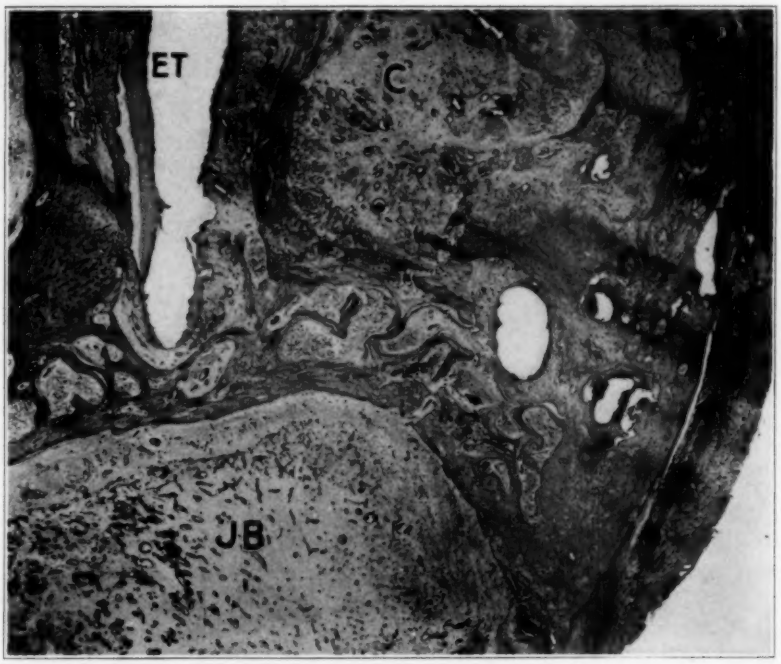


Fig. 9.—Vertical section through the cochlea and jugular bulb, showing the presence of healing stages of both labyrinthitis in the former and thrombophlebitis in the latter. *C* represents the cochlea, *ET* the eustachian tube and *JB* the jugular bulb.

auditory meatus and in the aquaeductus cochleae—was frequently observed. While these changes were most often secondary to an extension of the lesion in the sigmoid sinus and in the bulb, they were at times secondary to disease elsewhere in the temporal bone. The exact pathway of extension of the infection to the meninges, however, could not always be ascertained. A significant though relatively rare route of extension of infection from the sigmoid sinus is one by which it spreads

5. Druss, J. G.: Labyrinthitis and Sinus Thrombosis Complicating Suppuration of the Middle Ear, Arch. Otolaryng. **19**:671 (June) 1934.

along the dura of the posterior fossa to involve the saccus endolymphaticus, with its attendant reactive changes in the labyrinth. This mode of propagation to the labyrinth was suggested in 1 of the cases (case 5).

Reparative processes in sinus thrombosis apart from those associated with the early stages of the actual formation of the thrombus were evidenced by the presence of fibrosis and recanalization (fig. 10) within the veins and by formation of new bone adjacent to the wall of the vessel (fig. 4). These reparative processes were observed relatively early in the disease, that is, within two weeks after the onset of symptoms. Of significance is the fact that concomitantly with these healing

Fig. 10.—Process of recanalization in the inferior petrosal sinus. *IPS* represents the inferior petrosal sinus.

changes there frequently occurred evidence of the breaking down of the thrombus, with disintegration of the adjacent venous wall (fig. 2).

COMMENT

In the majority of instances thrombophlebitis of the sigmoid sinus is secondary to suppuration of the mastoid (fig. 11). The pathologic process in the bone can often be traced with the naked eye directly to the sigmoid sinus. At times, however, the disease in the bone cannot be followed grossly to the sigmoid sinus, the infection having reached the latter by way of microscopic venules. A perisinal abscess is more apt to occur with the former method of propagation than with the latter.

Thrombosis usually takes its origin at the site of a localized area of phlebitis. With necrosis of the intimal layer, a parietal thrombus is laid down. This gradually increases in size until it occupies the entire lumen, and an obturating thrombus is thus formed. The thrombus, on the other hand, may be parietal in character and organize and heal as such. The growth of the thrombus is most often found to be more pronounced in the direction of the blood current, that is, toward the heart, than in the opposite direction. Also, the size and extent of the thrombus need not correspond to the area of the adjacent phlebitis. The phlebitis usually extends for a considerable distance beyond the

Fig. 11.—Well organized thrombus in the sigmoid sinus. The inflammatory process in the mastoid, with necrosis of the sinus plate, also can be seen. The sinus in this case was implicated by direct extension. *M* represents the mastoid cavity, *SP* the sinus plate, *SS* the sigmoid sinus and *T* the thrombus.

limits of the thrombus. This phlebitic process is undoubtedly an important factor in the causation of continued bacteremia observed clinically after the thrombus has been removed at operation (Friesner[6]).

In a small proportion of cases the thrombophlebitis is produced as a result of the spread of infection not from the mastoid to the sigmoid

6. Friesner, I., in discussion on Ersner, M. S., and Myers, D.: Treatment of Thrombosis of Lateral Sinus Without Ligation of the Internal Jugular Vein, J. A. M. A. **109**:919 (Sept. 18) 1937.

sinus, as described, but rather from the middle ear directly to the jugular bulb. So-called primary thrombosis of the jugular bulb thus occurs. Naturally, the disease process in the bulb may, in the course of time. extend upward to implicate secondarily the sigmoid sinus.

Of further interest is the fact that the jugular bulb was seen to be diseased in virtually every case in which the sinus was thrombosed. This can be accounted for chiefly on an anatomic basis. Aside from the fact that the jugular bulb is the direct continuation of the sigmoid sinus, the latter makes a sharp curve forward and upward at its entrance to the former. As a result the slowing and the eddying of the blood flow at this site are factors conducive to thrombosis.

Extension to the jugular bulb may also take place from an infection in the petrous pyramid, namely, in the infralabyrinthine pneumatic cells. The formation of a peribulbar abscess as an intermediary stage is a not infrequent finding.

The superior and the inferior petrosal sinus are usually involved by retrograde extension from the sigmoid sinus or the jugular bulb. Occasionally, however, as is the case with the jugular bulb, previously mentioned, thrombosis in the petrosal sinuses is secondary to disease in the petrous pyramid, the infection advancing by means of bony necrosis, through fine capillaries or by a combination of these routes. In view of the relative frequency of thrombophlebitis of the superior and the inferior petrosal sinus, it is surprising that the cavernous sinus, which is the direct continuation of these vessels, is not more often similarly affected. It should be remembered, however, that engorgement and perivascular infiltration of the cavernous sinus is not an unusual histologic observation, the condition exhibiting few or no clinical manifestations.

The emissary vein also is usually thrombosed by retrograde extension from the sigmoid sinus, though there is sufficient evidence that thrombosis takes place by direct extension from an infection in the mastoid cells (Ziegelman [4]). The condition resulting from the latter method of propagation may be termed primary thrombosis of the emissary in contradistinction to secondary thrombosis of the emissary resulting from the former method. In a similar way the other venous tributaries of the sigmoid sinus and the jugular bulb, e. g., the vertebral and the anterior and the posterior condyloid veins,[7] are in all probability also affected.

Of significance is the observation in 1 of the cases of thrombosis localized to the jugular bulb in the presence of mastoiditis (case 15). At postmortem examination the sigmoid sinus was incised from the region of the jugular bulb to the end toward the torcular Herophili

7. These vessels, lying outside the domain of the blocks of the temporal bone as usually prepared, were not studied histologically.

and was seen to be relatively normal. The jugular vein also showed no abnormalities. The jugular bulb itself was not investigated at the time, and it was assumed that it also was free of disease. On histologic examination of the temporal bone, however, an organized thrombus limited to the jugular vault was disclosed (fig. 1). Though purulent exudate was present in the mastoid cells, the infection extended from the tympanum to the bulb by way of bony dehiscences. Three points are gleaned from this case: first, that primary thrombosis of the jugular bulb can take place even in the presence of mastoiditis; second, that at postmortem and histologic examination the venous sinuses must be carefully scrutinized before the final diagnosis of thrombosis is established, and, third, that localized thrombophlebitis in the jugular bulb may exist without being disclosed clinically at operation. As is well known, the jugular bulb in the usual routine operative procedure for sinus thrombosis is seldom if ever fully exposed or investigated. While it is frequently assumed that such a lesion exists, conclusive evidence of its existence after recovery of the patient is never really established.

That thrombosis of the sigmoid sinus may exist in the absence of clinical signs and symptoms is well known. In cases in which thrombosis was unsuspected it has not infrequently been discovered accidently at operation or at postmortem examination. Day [8] in 1915 reported 6 such cases, in which the condition was disclosed at operation, and in 4 of the 15 cases here reported it was accidentally discovered at postmortem or histologic examination. The difficulty in diagnosing thrombophlebitis clinically is due among other factors to the following facts: The thrombus may not be infected, or the infecting organism may be of such low virulence that characteristic signs and symptoms are not evoked. The clinical picture may be masked by signs and symptoms of a coexisting complication, e. g., acute meningitis or acute endocarditis (Rosenwasser [9]). The thrombophlebitic process may be a terminal event.

Of all the complications of sinus thrombosis, meningitis is the one most dreaded. This is brought about chiefly in three ways: (1) by extension through the blood stream, (2) by retrograde extension of the infection in the multiple tributaries which traverse the subarachnoid and subdural spaces (Nielsen and Courville [10]) and (3) by direct extension through the inner wall of the sinus. When the two condi-

8. Day, E. W.: Report of the Spontaneous Cure of Six Cases of Unrecognized Lateral Sinus Thrombosis Accidently Discovered During Operation, Laryngoscope **25**:757, 1915.

9. Rosenwasser, H.: Differential Diagnosis Between Thrombosis of the Lateral Sinus and Bacterial Endocarditis, Arch. Otolaryng. **26**:668 (Dec.) 1937.

10. Nielsen, J. M., and Courville, C. B.: Intracranial Complications of Otogenous Thrombosis of the Lateral Sinus, Ann. Otol., Rhin. & Laryng. **46**:13 (March) 1937.

tions coexist the meningitis is not always secondary to the sinus thrombosis per se. It may be secondary to direct invasion of the meninges by a continued infection within the structures of the temporal bone, the thrombosis of the sigmoid sinus being coincidental.

One is impressed with the observation, made by a study of the sections, of the not infrequent presence of an active suppurative process in the tympanomastoid area and in the petrous pyramid, occasionally even after a complete mastoidectomy (as a preliminary step to the operation on the sinus) has been performed. It is not the purpose of this paper to discuss the various factors responsible for this phenomenon. I am concerned here chiefly with the bearing that it has on the progress of the sinus thrombosis and the final outcome for the patient. There can be no question that the continued suppurative process in the bone, by exerting an unfavorable influence on the healing processes within the sigmoid sinus, may indirectly evoke terminal meningitis. It must therefore be regarded as a significant factor contributing to the prevalent high mortality from sinus thrombosis. On the basis of these observations the clinical importance of completely eradicating the original bony focus and the contiguous disease is self evident and cannot be overemphasized.

It must also be remembered that the formation of thrombus is actually nature's attempt to wall off the infection in the vein. While this attempt is successful in the majority of instances, in some, for one reason or another, the thrombus breaks down, with attendant dissemination of the infection. In the cases in which the processes of repair continue favorably, the thrombus acquires a fibrotic character but with an increase in the number and size of the vascular spaces. The newly formed bone adjacent to the vessel, observed particularly in the region of the jugular bulb, often exhibits a strong tendency to narrow the lumen. Recanalization within the vessel is the final stage.

TREATMENT

While it is true that the total number of cases studied is too small to permit even an attempt at drawing conclusions as regards surgical treatment, nevertheless certain histologic observations stand out, which have a bearing on this phase of the subject and which are worthy of consideration.

The jugular bulb was thrombosed in virtually every instance, both in the cases in which there had been an operation and in those in which there had been none. This indicates a strong tendency of the infectious process to advance in the direction of the blood current, that is, toward the heart. Consequently the basic principle involved in the act of ligating the jugular vein in the attempt to shut off the main avenue through which the infection enters the blood stream seems a

logical one. The fact that the infection may enter the circulation through the venous tributaries of the jugular bulb, that is, through the inferior petrosal sinus and the anterior and the posterior condyloid vein, by retrograde extension cannot be considered a strong argument against the advisability of using this procedure. It is true that these tributaries have frequently been seen to be thrombosed on histologic examination, but it is also true that this condition existed in the patient who had not been operated on as well as in the one who had. While involvement of these veins does not always elicit characteristic signs and symptoms and the condition is not often diagnosed clinically, it is my impression that these veins are not infrequently involved in patients who recover.

The reasoning which applies to the act of ligating the jugular vein is equally applicable to the procedure of occluding the sigmoid sinus in the region of the torcular Herophili, at a site beyond the apparent thrombophlebitis. Cognizance is taken of the fact that disease in the wall of the vessel beyond the site of obliteration is not infrequently observed histologically, but this does not militate against the validity of the principle.

Finally, the procedure of incising the sigmoid sinus, removing the infected thrombus and resecting the venous wall is basically sound. By this means it is hoped that a thrombus will be formed which is obturating in nature, which is relatively sterile and which will ultimately heal satisfactorily. Nature at times accomplishes these ends without operation, it is true, but because of the uncertainty it is more advisable to rely on the judgment and skill of the surgeon.

As is well known, there has been a diversity of opinion among the leading otologists, both here and abroad, concerning the surgical treatment of sinus thrombosis. This is particularly true as regards the indication for ligation of the jugular vein.[11] One school advocates this procedure in every case of sinus thrombosis; the other advocates its use only in selected instances, according to the clinical picture and the pathologic findings at operation. Until about five or six years ago the members of the otologic staff at Mount Sinai Hospital had rigidly adhered to the former school of thought; more recently however, we

11. Lillie, H. I.: Infection of the Sigmoid and Lateral Sinus, Surg., Gynec. & Obst. **35**:418 (Oct.) 1922. Mygind, S. H.: Treatment of Otogenic Sepsis and Sinus Thrombosis, Acta oto-laryng. **16**:474, 1931. Rott, O. M.: Why Ligate the Jugular in Cases of Lateral Sinus Thrombosis? Arch. Otolaryng. **14**:272 (Sept.) 1931. Coates, G. M.; Ersner, M. S., and Persky, A. H.: Lateral Sinus Thrombosis with a Review of the Literature, Ann. Otol., Rhin. & Laryng. **43**:419 (June) 1934. Meltzer, P. E.: Treatment of Thrombosis of Lateral Sinus: Summary of Results at Massachusetts Eye and Ear Infirmary, Arch. Otolaryng. **22**:131 (Aug.) 1935. Friesner.[6]

Case, Age, Sex	Duration of Aural Signs and Symptoms	Physical Signs and Symptoms	Signs and Symptoms of Intracranial Complication on Admission	Findings in Mastoid at Operation	Findings in Sinus at Operation; Operative Procedure	Secondary Operation on Sinus	Blood Culture
1 6 yr. M	2 wk.	Discharge from both ears; pain in both mastoids; temperature 104 F.; chronic discharge from ears for 2 years; enlarged cervical glands; cardiac signs	.....	Small acellular mastoid; for ward-lying sinus; mastoid free of disease	Sinus dull, lusterless, discolored; jugular vein collapsed; pus from jugular bulb; ligation of jugular vein	Free bleeding from region of torcular Herophili; pus expressed from jugular bulb	+ Str. haemolyticus
2 8 yr. F	3 wk.	Mastoidectomy in another institution; chills following; pus in urine; temperature 106 F.; drowsy and apathetic; postauricular discharge; palpable spleen; pneumonia at base of right lung; bilateral ankle clonus	Bilateral ankle clonus	Purulent exudate in many mastoid cells	Vein discolored, covered with granulations; obliterating purulent thrombus; no bleeding from bulb; emissary vein thrombosed; jugular ligation	No bleeding or pus in bulb; free bleeding from end toward torcular Herophili	+ Str. haemolyticus; Ottenb
3 12 yr. F	8 days	Aural pain and discharge; myringotomy; temperature 103 F.; headache; chilly sensation; attack of otitis in same ear 1 year before; drum full; serous discharge; mastoid tenderness	.....	Sclerotic mastoid; forward-lying sinus; pus in cells of tip; wall of sinus apparently normal	Obturating thrombus; no bleeding from superior petrosal sinus; jugular ligation; obliteration of sinus	.....	+ Str. haemolyticus; Ottenb
4 16 yr. M	2 mo.	Chronic discharge for 1 year; mastoidectomy and operation on sinus in another institution because of septic fever; thrombosis found; culture, Strep. anhaemolyticus; meningeal signs developed; stupor when admitted	Drowsiness; aphasia; choked disk; signs of temporosphenoidal abscess; facial paralysis; Babinski sign on left	.....	.....	.....	+ Str. haemolyticus
5 32 yr. M	8 days	Severe otalgia; aural discharge; headache; mastoid tenderness; sagging of wall of canal	Readmission; disorientation; aphasia; bilateral ophthalmoplegia; rigidity of neck; Babinski sign; lumbar puncture—fluid turbid; Pneumococcus type III	Peculiar reddish appearance of mastoid bone; no frank pus; necrotic in areas; culture of pus—Pneumococcus type III	.....	.....	+ Pn coccu type I short befor death
6 8 yr. F	9 days	Profuse aural discharge following protracted course of mild sepsis (?); signs of mastoiditis developed in hospital	.....	Subperiosteal abscess; necrosis of bone; large perisinal abscess; sigmoid sinus covered with granulations	.....	.....	.....
7 5 yr. F	6 wk.	Aural discharge for 6 weeks; temperature 102 F.; enlarged cervical glands; repeated chills	.....	Bone softening with little pus; perisinal abscess	Necrotic venous wall; obturating thrombus; no bleeding from jugular bulb; jugular ligation; obliteration of sinus	.....	+ Str. haem lytic
8 55 yr. M	4 wk.	Aural discharge for 4 weeks; vertigo; headache for 2 weeks; vomiting for 1 day; pulsating discharge from ear; tenderness over mastoid; nystagmus; markedly impaired hearing	Bilateral Kernig sign; rigidity of neck; bilateral Babinski sign; spinal fluid culture—Pneumococcus type III	Marked destruction of bone; large perisinal abscess	.....	.....	.....

Blood Culture	Metastases	Fundi	Duration of Illness After Operation on Sinus; Course in Hospital	Observations at Autopsy	Histologic Changes in Temporal Bone	Comment
+ Str. haemolyticus	Left wrist; right ankle	Normal	9 days; persistent signs of sepsis	Localized meningitis; acute ulcerative endocarditis; pericardial effusion; thrombosis of left superior petrosal sinus, occipital sinus and torcular Herophili	Purulent exudate in hypotympanum; purulent thrombus in sigmoid sinus, jugular bulb and superior and inferior petrosal sinuses	This is a case of primary thrombosis of the bulb secondary to an infection in the tympanum and eustachian tube
+ Str. haemolyticus; Ottenb	.....	Slight blurring	14 days; horizontal nystagmus; bilateral Kernig and Brudzinski signs; spinal fluid culture—Str. haemolyticus	Thrombosis of right lateral sinus extending to torcular Herophili, superior longitudinal and proximal portion of left lateral sinus; thrombosis of cerebellar veins; basilar meningitis	Sigmoid sinus partially occluded by organized thrombus; purulent in areas; beginning recanalization; intima necrotic; purulent thrombus in jugular bulb undergoing organization; thrombosis of superior petrosal sinus; hemorrhagic and serous labyrinthitis; active inflammation in peritubal cells	
+ Str. haemolyticus; Ottenb	.....	Blurring of margins of disk increased to papilledema 1.5 D	8 days; spinal tap prior to operation on sinus; culture—Str. haemolyticus; meningeal signs; nystagmus	Thrombosis of lateral sinus; acute diffuse purulent meningitis	Cholesteatoma in antrum in contact with dura; necrosis of jugular dome; organized thrombosis in sigmoid sinus; thrombophlebitis in jugular bulb; direct extension from hypotympanum; thrombosis of inferior and superior petrosal sinuses	
+ Str. haemolyticus	.....	Blurring of margins of disk advancing	6 weeks; exploratory operation for abscess of brain with negative findings; reexploration on 2 occasions, abscess found	Subdural abscess of left temporal lobe; purulent pachymeningitis; thrombosis of left lateral sinus; abscess of temporal lobe	Purulent exudate in petrous pyramid; dura over roof of antrum markedly inflamed; organized fibrous thrombus in sigmoid sinus and jugular bulb; evidence of healing processes in thrombus; thrombosis of superior and inferior petrosal sinuses; involvement of ninth and tenth nerves	Patient entered with signs of abscess of brain, which went on to bacterial meningitis in spite of operation
+ Pneumococcus type I; shortly before death	.....	.....	Nystagmus; total loss of hearing	Large extradural abscess on posterior surface of petrous pyramid; subacute bacterial meningitis	Suppuration in petrous pyramid; suppurative healing labyrinthitis; organized thrombus in lateral sinus and jugular bulb; large peribulbar abscess; thrombosis of superior petrosal sinus; meningitis	No clinical evidence of sinus thrombosis; multiplicity of lesions in temporal bone is of interest
.....	.....	.....	Nystagmus; signs of bronchopneumonia; meningeal signs	Acute thromboendocarditis; bronchopneumonia with abscesses; infarcts of kidney	Cholesteatoma in antrum; suppuration of petrous pyramid; thrombophlebitis of sigmoid sinus and jugular bulb; peribulbar abscess	Thrombosis of sigmoid sinus present without clinical evidence; jugular bulb probable site of involvement (primary)
+ Str. haemolyticus	.....	.....	Sepsis continued after operation on sinus; pus expressed from bulb; repeated transfusions	Acute diffuse purulent meningitis	Suppuration in petrous pyramid; serous labyrinthitis; communication of infection in tympanum with jugular bulb through dehiscences and blood vessels; purulent thrombophlebitis of sigmoid sinus and jugular bulb; necrosis of wall of vessel; thrombosis of inferior petrosal sinus extending to apex of petrosa	
.....	.....	.....	Caloric test showed dead labyrinth; ceased 4 days after admission	Diffuse purulent meningitis	Suppuration in petrous pyramid; suppuration in labyrinth; fistula in posterior semicanal; large peribulbar abscess; purulent thrombophlebitis of sigmoid sinus, jugular bulb and superior and inferior petrosal sinuses	Thrombosis of sigmoid sinus disclosed histologically in absence of clinical signs; multiplicity of lesions in temporal bone; patient entered hospital with signs of bacterial meningitis

Case, Age, Sex	Duration of Aural Signs and Symptoms	Physical Signs and Symptoms	Signs and Symptoms of Intracranial Complication on Admission	Findings in Mastoid at Operation	Findings in Sinus at Operation; Operative Procedure	Secondary Operation on Sinus	Blood Cultu...
9 2 mo. F	2 days after admission	Discharge from right eye since birth; swelling about right eye for 5 days; profuse discharge from nose; septic fever; right eye swollen, indurated; appears toxic; discharge from right middle ear 2 days after admission	Signs of early cavernous sinus thrombosis	.....	.....	.....	..... Metas... tases
10 36 yr. M	7 wk.	Aural discharge for 7 weeks; frontoparietal headache and pain on side of face for 3 weeks; chill 2 weeks before; feverish; discharge from same ear 16 years before; profuse foul-smelling discharge; marginal perforation; sagging; impaired hearing	Mental confusion; aphasia; projectile vomiting; abscess of brain (?) in left temporal lobe	Extensive disease in mastoid and in petrosa; large perisinal abscess	.....	.....	.....
11 8 yr. F	4 wk.	Otalgia 4 weeks before; myringotomy but no discharge; otalgia ceased in 4 days; chills; high fever; pains in joints; drum congested but otherwise normal; enlarged cervical glands; liver and spleen palpable; metastases to joints	Stupor; vomiting of blood; suppression of urine	.....	.....	.....	+ Str... haem... lytic... ...t knee ...elbow; ...t knee; ...er part ...spine
12 46 yr. F	13 days	Otalgia for preceding 2 weeks; noises in ear; dizzy sensation in preceding month; discharging ear for 36 years; diminished hearing; huge polyp in external canal; foul-smelling discharge; hearing markedly impaired	Labyrinthine signs	Radical mastoidectomy; cholesteatoma filling antrum; granuloma filling antrum; Panse flap	Plastic exudate on sigmoid sinus in region of bulb; intima of vessel thickened; little bleeding from bulbar region; jugular ligation; obliteration of sinus	.....	+ Str... haem... lytic...
13 3 yr. F	4 wk.	Otalgia; myringotomy; profuse discharge; mastoid thickening and tenderness	.....	Extensive disease in mastoid; sinus inadvertantly exposed, found normal	Sinus covered by granulations; phlebitis but free bleeding from sinus; jugular ligation; obliteration of sinus	Frank pus expressed from bulb; free bleeding from end toward torcular Herophili	+ Str... haem... lytic...
14 6 yr. M	5 wk.	Otalgia 5 weeks before; recurrence 2 weeks before; myringotomy; convulsion 10 days before; chill 6 days before; septic fever with temperature 99 to 105 F. daily for preceding 5 days; drum flat; landmarks visible; scant discharge; palpable spleen	.....	Necrosis of bone; thick pus in mastoid; wall of sinus apparently normal on exposure	Jugular vein grayish white, collapsed; jugular ligation; obliteration of sinus	.....	+ Str... haem... lytic...
15 48 yr. M	16 days	Otalgia 16 days before; chills 15 days before; septic fever with temperature to 105 F.; frontal headache for 5 days; generalized rash; drum relatively normal; petechial hemorrhages (white); systolic murmur at apex	Drowsiness; rigid neck; bilateral Kernig sign; absence of abdominal reflexes; spinal puncture—yellow, turbid fluid, 2,700 cells	.....	.....	.....	+ Pn... cocc... type ... Otte...

Blood Culture	Metastases	Fundi	Duration of Illness After Operation on Sinus; Course in Hospital	Observations at Autopsy	Histologic Changes in Temporal Bone	Comment
.....	.....	.....	Chemosis of both eyelids; proptosis of right eye; ceased 3 days after admission	Purulent cavernous sinus thrombosis; purulent pachymeningitis; periostitis of right maxilla	Suppurative process in tympanum, mastoid and petrosal cells; large peribulbar abscess; purulent thrombosis of sigmoid sinus, jugular bulb and superior and inferior petrosal sinuses; thrombosis of cavernous sinus; necrosis of carotid canal; partial necrosis of carotid artery; ninth and tenth nerves bathed in pus	The sigmoid sinus and jugular bulb involved secondary to cavernous sinus thrombosis by way of petrosal sinuses
.....	.....	Blurring of disks	Exploratory operation for abscess of brain; pus evacuated; revision two times, pus evacuated each time	Abscess of the temporal lobe; meningoencephalitis; parenchymatous degeneration of heart	Cholesteatoma invading attic and antrum; suppuration in petrous pyramid; organized thrombus in sigmoid sinus but partially necrotic in areas	Patient entered hospital with signs of abscess of temporal lobe; there was little if any clinical evidence of sepsis; it was masked by signs of abscess of brain
+ Str... haem... lytic...	...ht knee; ...t elbow; ...t knee; ...er part ...spine	.....	Child lived for 1 day after admission	Thrombosis of jugular bulb; hemorrhagic infarcts to both kidneys; subacute infectious splenic swelling; hypertrophy of right ventricle	Mild inflammatory changes in tympanum and mastoid; purulent thrombophlebitis localized to jugular bulb; thrombosis of inferior petrosal sinus; thrombophlebitis of vessels in jugular dome	Case of primary thrombosis in the bulb in which the sigmoid sinus was uninvolved; scant aural signs; patient in stuporous condition on admission; no operation
+ Str... haem... lytic...	.....	Indistinctness of nasal margins	4 days; signs of intracranial inflammatory lesion appeared after radical mastoidectomy; signs of meningitis appeared shortly after operation on sinus	Diffuse purulent leptomeningitis; encephalorrhagia	Cholesteatoma of tympanum and aditus; fistula of external semicanal and erosion of fallopian canal; acute suppurative labyrinthitis; inflammation in petrous pyramid; thrombophlebitis of jugular bulb; meningitis	Jugular bulb probably initial site of thrombophlebitis; presence of cholesteatoma and suppurative labyrinthitis were contributing factors in causing fatal issue
+ Str... haem... lytic...	.....	.....	Sudden rise in temperature 6 days after operation; septic fever continued after first operation on sinus	Thrombophlebitis of sigmoid sinus; parenchymatous degeneration of heart, liver and kidneys	Purulent thrombophlebitis of sigmoid sinus, jugular bulb and superior petrosal sinus; dehiscences in jugular dome	
+ Str... haem... lytic...	.....	Bilateral optic atrophy	Convulsions following operation; ceased 17 hours after operation	.....	Purulent exudate in petrous pyramid; purulent thrombus in sigmoid sinus; jugular bulb and inferior petrosal sinus; inflammation about ninth and tenth nerves; thrombosis in veins in floor of hypotympanum communicating with jugular bulb	Patient extremely ill on admission; jugular bulb probable site of initial involvement; child did not rally after operation
+ Pn... cocc... type... Otte...	.....	.....	Meningeal signs increased in severity; ceased 3 days after admission	Acute bacterial endocarditis; myocardial abscesses; embolic abscesses in lung; acute purulent meningitis; sigmoid sinus free of disease	Purulent exudate in tympanum and mastoid; thrombophlebitis limited chiefly to jugular bulb through multiple dehiscences in jugular dome through which infection spread; jugular bulb high; sigmoid sinus forward lying; no evidence of disease in sigmoid sinus	Case demonstrates primary thrombosis in the bulb in presence of mastoiditis; infection spread through dehiscences in jugular dome; presence of white-centered petechiae and endocarditis masked clinical picture

have on occasion deviated from it. At the present time it is still my opinion that the basic principle implied in the surgical treatment of sinus thrombosis, i. e., eradicating the focus in the bone and in the sigmoid sinus and attempting to seal off the infection from the blood stream by ligation of the jugular vein and by obliteration of the sinus beyond the apparent site of disease in the vein, is fundamentally sound. But I feel that at times it is advisable to modify or omit one or all of the steps. This view is based not only on a study of the 15 cases herein described but on a review of the literature and past clinical experience.

SUMMARY AND CONCLUSIONS

An analytic study, including a report of the histologic sectioning of the temporal bone, is made of 15 cases of proved thrombophlebitis of the sigmoid sinus or jugular bulb. The pathologic processes in the temporal bone and the routes of extension to the various venous sinuses within the structure are particularly emphasized.

Thrombophlebitis of the sigmoid sinus, unrecognized during life, may be accidentally disclosed at autopsy or by histologic examination of the temporal bone.

The jugular bulb is frequently the seat of involvement. It was diseased in each of the 15 cases studied; its infection is usually secondary to extension from the sigmoid sinus. Extension to the jugular bulb also takes place directly from the tympanum, when it is known as primary thrombosis of the bulb.

Thrombophlebitis localized to the jugular bulb (primary thrombosis of the bulb) may exist in the presence of mastoiditis.

Thrombosis of the superior and the inferior petrosal sinus and other venous tributaries is not infrequently observed on histologic examination. These vessels may act as pathways of infection for meningitis, and their involvement may be a contributing factor to the high mortality from sinus thrombosis.

The necessity for careful investigation of all venous sinuses before establishing a final diagnosis is emphasized.

Meningitis when present may not be due directly to the sinus thrombosis per se; it may be secondary to an intercurrent infection in the temporal bone, e. g., in the petrous pyramid or the labyrinth. The clinical importance of completely eradicating all disease in the bone is stressed.

In the surgical treatment of sinus thrombosis I feel that the classic operation advocated in 1884 by Zaufal and soon after by Ballance and McCewen, which includes ligation of the internal jugular vein, incision and removal of infection in the sinus and obliteration of the latter, is still, except on rare occasions, the procedure of choice.

INCIDENCE OF MALIGNANT TUMORS OF THE HEAD AND NECK

A STATISTICAL SURVEY

NOAH D. FABRICANT, M.D.
CHICAGO

In the seven years from July 1, 1930, to July 1, 1937, 249 cases of malignant tumor about the head and neck were recorded in the Department of Otology, Rhinology and Laryngology of the University of Illinois College of Medicine. The tumors were representative of the types of malignant growth found about the head and neck. Allowing for the advent of a period such as 1932-1933, during which an economic crisis deepens and projects an increasing number of patients into clinics and dispensaries, the number of new patients admitted each year to the department is fairly constant and totals approximately 2,500 per annum.

Year	New Patients
July 1, 1930 to Jan. 1, 1931	1,123
Jan. 1, 1931 to Jan. 1, 1932	2,708
Jan. 1, 1932 to Jan. 1, 1933	3,217
Jan. 1, 1933 to Jan. 1, 1934	2,872
Jan. 1, 1934 to Jan. 1, 1935	2,384
Jan. 1, 1935 to Jan. 1, 1936	2,446
Jan. 1, 1936 to Jan. 1, 1937	2,304
Jan. 1, 1937 to July 1, 1937	1,330

Although more than 249 cases of malignant growth have been observed at the University of Illinois College of Medicine during the seven year period, only that number will be considered here, since for the purposes of this survey no patient has been regarded as having a malignant condition unless a histologic study had been made at the institution and a diagnosis of a neoplastic process confirmed microscopically. I particularly wish to emphasize this point, because too often in the past statistics concerning malignant growths have been based on clinical interpretation. During the seven years a total of 1,103 biopsies were made by various members of the department; of these biopsies 249 were regarded as indicating a malignant tumor.

Read before the Chicago Laryngological and Otological Society, March 7, 1938.

From the Department of Otology, Rhinology and Laryngology, Research and Educational Hospitals, University of Illinois College of Medicine.

Early in the current decade a small number of malignant growths were seen. Nine were detected during the second half of 1930. As the march of the nineteen-thirties progressed, more malignant tumors were seen. Thus, there were 30 in 1931, 25 in 1932, 20 in 1933, 33 in 1934, 48 in 1935, 55 in 1936 and 29 in the first six months of 1937. Several years ago a departmental policy of dispensing with the routine biopsy of nasal polypi and chronically infected tonsils was adopted. By establishing a ratio of the number of biopsies indicating neoplasm to the total number of biopsies, it was learned that 15.5 per cent of the biopsies indicated malignant growth in 1930, 17.2 per cent in 1931, 10.3 per cent in 1932, 12.3 per cent in 1933, 25.7 per cent in 1934, 26.8 per cent in 1935 and 34.1 per cent in 1936.

Although the number of new patients admitted yearly to the department has been rather constant, the incidence of malignant growth among the new patients has been more than doubled within seven years. From less than 1 per cent it has increased to more than 2 per cent.

Year	Incidence, Percentage
July 1, 1930 to Jan. 1, 1931	0.8
Jan. 1, 1931 to Jan. 1, 1932	1.1
Jan. 1, 1932 to Jan. 1, 1933	0.7
Jan. 1, 1933 to Jan. 1, 1934	0.6
Jan. 1, 1934 to Jan. 1, 1935	1.3
Jan. 1, 1935 to Jan. 1, 1936	1.9
Jan. 1, 1936 to Jan. 1, 1937	2.3
Jan. 1, 1937 to July 1, 1937	2.1

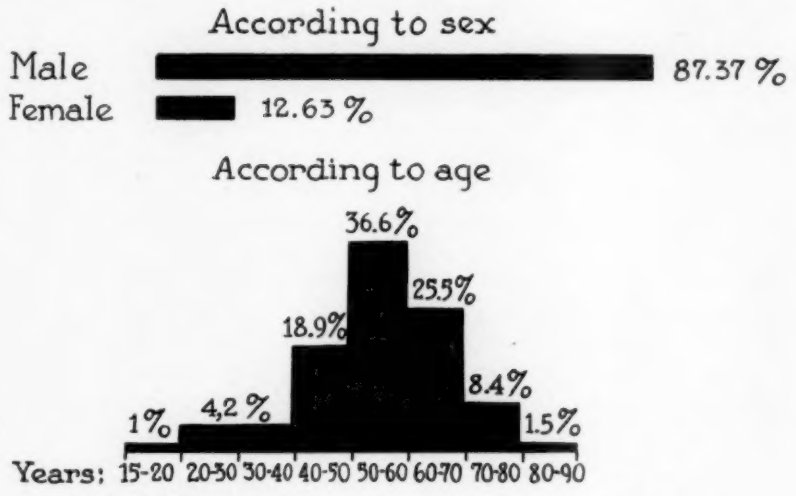
Since 1935 the department has had the services of a bronchoscopist, Dr. Paul Holinger, who occupies himself with malignant tumors about the esophagus and the bronchial tree. Because of his preoccupation there has been an increase in the number of patients with esophageal or bronchial tumor coming to the University of Illinois College of Medicine, and this is reflected in the statistics.

The youngest patient covered in the survey was a boy 17 years old; the next youngest, a girl 19 years old. The oldest patient with a malignant tumor was a man 85 years old; the next oldest, a woman 84 years of age. In determining the distribution of malignant tumors according to sex, I found that 87.37 per cent of the total existed in men and but 12.63 per cent in women. In other words, malignant tumors occurred more than seven times as frequently in men as in women. These percentages, it occurs to me, are noteworthy, since in an adjacent institution, the Cook County Hospital, Boot[1] analyzed a series of 250 malignant tumors about the head and found 88.4 per cent in men and 11.6 per cent in women.

1. Boot, G. W.: Cancer in Eye, Ear, Nose and Throat Practice, Illinois M. J. **56**:349-354 (Nov.) 1929.

The incidence of malignant tumors according to age reveals an appreciable rise during the fourth decade of life, the attainment of a peak during the fifth decade and a gradual decline during the sixth decade. It is interesting to observe that most investigators have found the fifth decade to be the period in which the largest number of malignant tumors of the head and neck develop, whereas the fourth and sixth decades appear interchangeably in second place. The chart graphically illustrates the distribution of malignant tumors according to sex and age.

In the present series of 249 cases of malignant tumor, 78 patients had Wassermann and Kahn tests, and only 2 had a positive (4 plus) reaction. For 6 patients who underwent tests for tuberculosis the results were negative.



Distribution of malignant tumors according to sex and age.

The following tabulation represents the anatomic site, the type of tumor and the number of cases encountered in the survey:

Anatomic Site	Type of Tumor	No. of Cases
1. Larynx	Squamous cell carcinoma	54
	Undifferentiated carcinoma	1
	Epidermoid carcinoma	2
		57
2. Tongue	Squamous cell carcinoma	22
3. Esophagus	Squamous cell carcinoma	28
	Adenocarcinoma	1
		29

Anatomic Site	Type of Tumor	No. of Cases
4. Neck	Squamous cell carcinoma	10
	Epidermoid carcinoma	1
	Transitional cell carcinoma	2
	Medullary cell carcinoma	1
	Lymphoma	1
	Lymphosarcoma	1
	Plasmacellular granuloma	1
	Lymphoblastoma	1
		18
5. Tonsil	Squamous cell carcinoma	9
	Leukosarcoma	1
		10
6. Bronchus	Squamous cell carcinoma	11
	Undifferentiated carcinoma	1
	Transitional cell carcinoma	3
	Adenocarcinoma	2
	Medullary carcinoma	1
	Bronchogenic colloid carcinoma	1
	Alveolar carcinoma	1
		20
7. Epiglottis	Squamous cell carcinoma	6
8. Nose	Squamous cell carcinoma	5
	Basal cell carcinoma	7
	Undifferentiated carcinoma	1
	Transitional cell carcinoma	2
	Adenocarcinoma	1
	Medullary carcinoma	1
	Round cell carcinoma	2
	Lymphoma	1
	Mixed sarcoma and carcinoma	1
	Angiosarcoma	1
	Cylindric cell carcinoma	1
		23
9. Mouth	Squamous cell carcinoma	3
	Basal cell carcinoma	1
	Round cell carcinoma	1
	Lymphosarcoma	1
	Hodgkin's carcinoma	1
		7
10. Pharynx	Squamous cell carcinoma	7
	Undifferentiated carcinoma	1
		8

Anatomic Site	Type of Tumor	No. of Cases
11. Jaw	Squamous cell carcinoma	4
	Adenocarcinoma	1
		5
12. Ear	Squamous cell carcinoma	3
	Basal cell carcinoma	2
	Medullary carcinoma	1
	Carcinoma simplex	1
	Epidermoid carcinoma	1
		8
13. Thyroid	Squamous cell carcinoma	1
14. Antrum	Squamous cell carcinoma	1
	Undifferentiated carcinoma	2
	Epidermoid carcinoma	2
	Carcinoma simplex	1
	Sarcoma	1
		7
15. Cheek	Squamous cell carcinoma	2
16. Face	Squamous cell carcinoma	1
	Basal cell carcinoma	3
	Spindle cell sarcoma	1
		5
17. Lip	Squamous cell carcinoma	2
18. Gingiva	Squamous cell carcinoma	3
	Basal cell carcinoma	1
		4
19. Lung	Squamous cell carcinoma	2
	Undifferentiated carcinoma	1
		3
20. Eye and orbit	Basal cell carcinoma	7
	Mixed squamous and basal cell carcinoma	1
		8
21. Frontal sinus	Basal cell carcinoma	1
22. Submaxillary gland	Adenocarcinoma	1
23. Parotid gland	Mixed tumor suggesting carcinoma	1
24. Trachea	Squamous cell carcinoma	1
	Total	249

As is readily discerned, the largest number of malignant tumors invaded the larynx. Second in numerical significance are the tumors which invaded the nose and paranasal sinuses. Following closely are the invaders of the esophagus, the tongue, the bronchi and the tonsils. Seventy per cent of the 249 cancers are listed as being of the squamous cell type; 8.7 per cent were basal cell carcinomas, found chiefly about the eye, orbit and nose; 2.7 per cent were transitional cell carcinomas; 2.7 per cent were undifferentiated tumors, and 2 per cent were adenocarcinomas. The remaining types, including all the sarcomas, individually form less than 1 per cent of the total. The heavy predominance of squamous cell carcinoma is manifested by the fact that 95 per cent of the tumors invading the larynx, almost 97 per cent of those invading the esophagus, 100 per cent of those invading the tongue, 55 per cent of those invading the bronchi and the neck and 90 per cent of those invading the tonsils were of this type. Other favorite sites for squamous cell carcinoma, as revealed by the survey, are the pharynx, the jaw and, to a less extent, the mouth.

CONCLUSIONS

A statistical survey of the incidence of malignant tumors about the head and neck as seen at the University of Illinois College of Medicine during a seven year period (1930 to 1937), in which 249 cases were studied, establishes the following facts:

1. Malignant tumors occur 7 times more frequently in men than they do in women.

2. Bronchoscopy and esophagoscopy are important in enhancing and illuminating statistics of malignant tumors.

3. In each sex, approximately the same percentage of malignant tumors occurs in extreme youth and in extreme old age.

4. Most malignant tumors occur during the fifth decade of life, with the sixth and the fourth decades following in order.

5. The vast majority of tumors are of the squamous cell type.

185 North Wabash Avenue.

LARYNGEAL STENOSIS IN CHILDREN

WITH SPECIAL REFERENCE TO TREATMENT WITH CORE MOLDS

ELLEN J. PATTERSON, M.D.
PITTSBURGH

Laryngeal stenosis may be considered as acute or chronic, depending on the cause and the duration of symptoms. Any condition that narrows the lumen of the larynx and subglottic area in a relatively short time will cause laryngeal symptoms of acute stenosis, while symptoms in chronic stenosis may be insidious, developing over a longer period.

Laryngeal symptoms, whether acute or chronic, when encountered in children differ in many respects from laryngeal symptoms in adults, for several reasons; in fact, the symptoms may differ in the same disease in children and adults. The cartilages and general structure of a child's larynx are extremely delicate, and the glottic chink in a normally developed larynx is small and readily encroached on by a slight amount of inflammation or swelling of the laryngeal mucosa, a condition which interferes with respiration. The subglottic area in children under 3 years of age is vascular and the connective tissue loose; hence swelling quickly results from inflammation, infection or trauma and by mechanically shutting off the airway quickly becomes an important factor in all cases of laryngeal disturbance. An important point to be kept in mind in cases of acute infection causing edema is that the site of narrowing in children is in the conus elasticus.

Acute stenosis may be caused by the presence of a foreign body, by accumulation of secretion or exudate or by distention of the tissues by inflammatory products, such as serum or pus. Probably the most frequent of acute infections are diphtheria and acute laryngotracheitis due to streptococci, influenza bacilli or other organisms. Diphtheria may occur as a disease of the subglottic area without involvement of the upper portion of the larynx or pharynx, with the exudate causing acute stenosis. Acute laryngotracheitis, with its onset of alarming and sometimes fulminating symptoms of acute stenosis due to inflammation, edema and secretions, is a familiar picture to every physician.

When either prolonged intubation or emergency tracheotomy is necessary for the relief of acute stenosis, chronic stenosis frequently follows, especially in the case of a child. Laryngeal stenosis in the newborn may be due to various anomalies of the larynx or trachea or to

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Read at a meeting on the Seventh Pan-American Cruise Congress, Habana, Cuba, Jan. 21, 1938.

traumatism of these structures during delivery. Chronic laryngeal stenosis in children may be congenital or, like the proverbial cloak, may cover a multitude of sins, chief of which is faulty tracheotomy. Jackson and Jackson [1] stated that in 80 per cent of cases chronic laryngeal stenosis in children is due to improperly performed tracheotomy, especially to the high operation. Thomson [2] stated that badly executed tracheotomy is the most frequent cause of chronic laryngeal stenosis.

Laryngeal stenosis present in a child since birth may be regarded as congenital; the designation includes laryngeal stridor due to the exaggerated infantile type of larynx. In this condition the upper aperture of the larynx, from failure to develop properly, is abnormally plastic and has a tendency to collapse, which causes epiglottic deformity and an inspiratory crow. This type of larynx is observed in rachitic or underdeveloped children and in those who are artificially fed. The condition is usually relieved by improvement in nutrition. Congenital cyst of the larynx may cause symptoms of chronic stenosis or alarming symptoms of impending asphyxia, largely according to the size and location of the cyst. Previous to the advent of direct laryngoscopy, congenital cyst of the larynx was mistakenly diagnosed as enlarged thymus. Congenital web of the larynx is regarded by many writers as extremely rare, probably because of the dearth of cases reported in the literature, but Jackson and Jackson [3] observed that the "statements as to rarity are based on the records in the days when the examination of a living child's larynx was impossible." A congenital web may noticeably affect the cry of a baby, or it may give no evidence of its presence until the child begins to talk, when a defect in speech is observed. Larger webs produce symptoms noticeable as soon as respiration starts in a newborn child. Papilloma of the larynx is a frequent cause of chronic stenosis in children and may even occur in the newborn. It is usually multiple, has a tendency to repullulate and may even invade the trachea. A nonobstructive foreign body, when the larynx has become tolerant of its presence, may be the cause of chronic laryngeal stenosis.

Acute laryngeal stenosis in children with urgent laryngeal dyspnea requires intubation or tracheotomy for immediate relief. Except in cases of diphtheria, in which the intubation tube is usually dispensed with in a few days, tracheotomy is preferable for the relief of laryngeal dyspnea. The prolonged sojourn of an intubation tube in the delicate laryngeal structure of a child, with frequent reintroduction, has a

1. Jackson, C., and Jackson, C. L.: The Larynx and Its Diseases, Philadelphia, W. B. Saunders Company, 1937, p. 183.

2. Thomson, St. C.: Permanent Tracheostomy in Stenosis of the Larynx, J. Laryng. & Otol. **46**:817 (Dec.) 1931.

3. Jackson and Jackson,[1] p. 66.

tendency to cause stenosis, which ultimately requires tracheotomy. Tracheotomy is one of the oldest of surgical procedures, yet it is the one most frequently done improperly. It is generally undertaken with reluctance and frequently delayed until its execution becomes difficult; often its performance is unsatisfactory and the after-care mismanaged. There are no contraindications to tracheotomy for obstructive laryngeal dyspnea. When tracheotomy is properly performed, complications are rare and stenosis of the larynx will never result directly. To my mind there is no laryngologic operation the performance of which is attended by a thrill equal to that which one derives from the gratitude expressed in the eyes of a child who previously has been gasping for air, as he lies relieved and peaceful after tracheotomy. Frequently he falls asleep before being removed from the operating table.

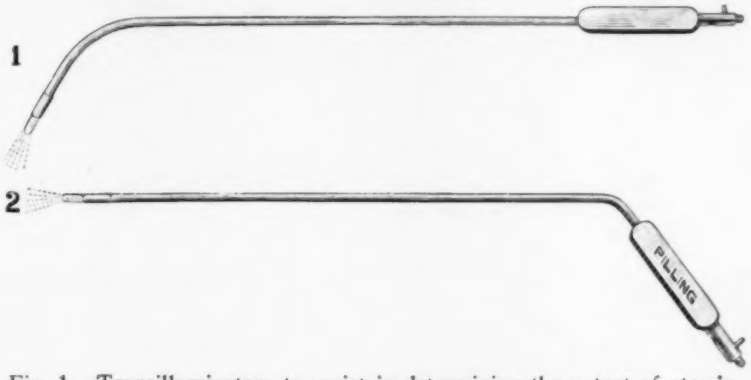


Fig. 1.—Transilluminators to assist in determining the extent of atresia.

No general anesthetic should be administered for tracheotomy, because any patient dyspneic enough to need tracheotomy is depending largely on the action of the accessory muscles of respiration, and if the action of these muscles is stopped by beginning unconsciousness, respiration ceases. The terms high and low, as used with reference to tracheotomy, should be eliminated, as these designations are related to the isthmus of the thyroid. Tracheotomy should always be low in order to avoid cutting the cricoid cartilage and traumatizing the subglottic area. Thus the possibility of chronic stenosis of the larynx is avoided.

The foregoing comment concerns the cause, the pathologic structure and prophylaxis. Treatment remains to be considered.

It is paradoxic that, though in most cases stenosis is the result of tracheotomy, yet the first step in its correction of a stricture is repetition of the procedure. A correctly executed opening in the trachea is essential. A preliminary requisite to treating chronic laryngeal stenosis in a child is to make his acquaintance and get his confidence; one then

decides on a plan of treatment, which sometimes requires years of laborious study. The plan of treatment is determined by the cause of the stenosis and by the information obtained from direct laryngoscopic examination and from study of roentgenograms of the larynx. Palpation of the neck is important, though often neglected. It assists in determining the condition of the laryngeal cartilages. In cases of complete atresia transillumination is an aid in determining its extent.

Low tracheotomy is the first step in treatment in cases in which the tracheal cannula is high or in an improper position, in the larynx instead of the trachea. It is important in making the incision for low

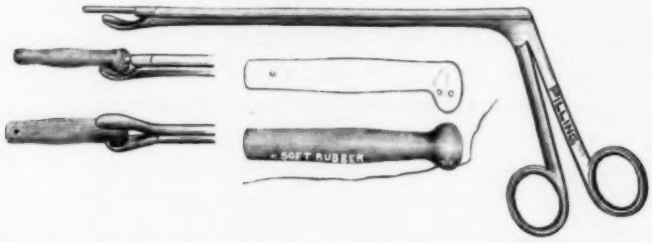


Fig. 2.—Core molds and forceps for removal of core molds.

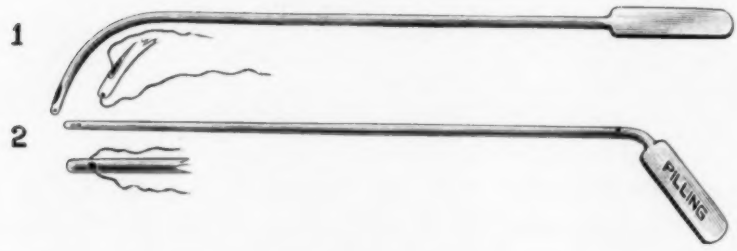


Fig. 3.—Carriers for introduction of thread through the larynx.

tracheotomy to leave a bridge of intact skin and tissue between it and the tracheal fistula; if this procedure is not followed, the cannula will ride up and occupy the old fistula. With the removal of the pressure of the tracheal cannula from the subglottic area in cases in which tracheotomy has recently been performed, stenosis sometimes clears up and decannulation is possible. In some cases relief can be obtained by direct laryngoscopic dilation with graduated laryngeal dilators, while in others a long period of treatment with elastic rubber dilators introduced into the larynx is required.

The treatment which I wish to emphasize is that with the rubber core molds devised by Chevalier Jackson. The soft rubber mold is

most effective in softening and absorbing obstructive cicatricial tissue. In the treatment of stenosis of the larynx in children, Jackson stated two fundamentals should be kept in mind; one is the growth of the child, and the other is the corking of the cannula. The larynx will grow if the child breathes through it, and in order to force the breathing the cannula must be corked.

REPORT OF CASES

At the present time there are 3 children under treatment for chronic laryngeal stenosis in the bronchoscopic clinic at the Presbyterian Hospital.

Fig. 4 (case 1).—Core mold no. 30 F in the larynx.

CASE 1.—Walter had an emergency tracheotomy at 8 months of age, probably for diphtheria. At 5 years of age he was brought to the bronchoscopic clinic by the Red Cross nurse for decannulation. At that time there was diaphragmatic stenosis in the subglottic region, with a central opening 1 mm. in diameter. After low tracheotomy, the weblike diaphragm was incised and direct laryngoscopic dilation carried out over a period of time, with satisfactory results while the patient was being treated. One and one-half years ago laryngostomy was performed and a no. 18 F rubber obturator anchored in the larynx with silver wire for five weeks. This treatment appeared satisfactory for a time. One year ago dilation was begun by means of elastic core molds; a no. 14 F obturator

was used at first and the treatment continued for six months, until it was possible to introduce a no. 30 F instrument (fig. 4). It has now been six months since the last core mold was removed. The patient is wearing a full cork in his tracheal cannula continuously for a test period and has a good voice.

CASE 2.—Bertha, aged 3 years, had twelve intubations for diphtheria followed by an emergency tracheotomy performed at home. Two months later she was brought to the Children's Hospital because of unrelieved dyspnea. The tracheal cannula was in the larynx instead of the trachea (fig. 5), and low tracheotomy

Fig. 5 (case 2).—The arrow shows the area of complete atresia of the larynx.

relieved the difficulty in breathing. The larynx was full of granulation tissue, which resulted in complete atresia of the larynx with absolute loss of voice. Direct laryngoscopic dilation was of no avail. Seven months ago the stenosed larynx was perforated and a no. 12 core mold introduced. After two months' treatment the child was discharged, and twenty days after discharge her father noticed that she had some voice. She has now reached the test period, has worn her tracheal cannula with full cork for two months and has a loud, clear voice. The glottis admits a 4 mm. bronchoscope easily.

CASE 3.—Clara had an emergency tracheotomy at 3½ years of age, probably for diphtheria. She was brought to the clinic by the Red Cross nurse at 9 years of age for decannulation. She had complete atresia of the larynx at the cricoid level (fig. 6), and after low tracheotomy direct laryngoscopic dilation was attempted, without results. Eighteen months ago laryngostomy was carried out and a rubber dilator anchored in the larynx with silver wire, without satisfactory results. One year ago the stenosed larynx was perforated and a no. 12 core mold introduced. This treatment was continued, the size of the core molds being increased until the patient was wearing a no. 28. After five months' absence she returned for further treatment and is now wearing a no. 28 core mold. She has developed a remarkable buccal speech.

Fig. 6 (case 3).—Complete atresia of the larynx.

COMMENT

These cases illustrate chronic laryngeal stenosis due to improperly performed tracheotomy in children and the great amount of labor necessary to effect cure.

The elastic rubber core molds, because of their shape and expansile pressure on the stenosed tissue, have given the best results.

121 University Place.

LINGUAL THYROID

CARROLL SMITH, M.D.
SPOKANE, WASH.

In the early months of fetal life, what is to be the thyroid gland is located at the base of the tongue in a depression, the foramen caecum, the upper end of the thyroglossal duct. Normally it descends along the duct to its position in front of the cricoid cartilage and the upper tracheal rings, where the duct ends.

Rarely this descent is interfered with, and the gland develops in whole or in part at the foramen caecum, forming a lingual thyroid. Still more rarely some of the cells lodge in or at the bottom of the tongue, and an intralingual or sublingual thyroid results. An accessory thyroid also sometimes occurs in front of the larynx, forming a prelaryngeal thyroid. Then comes the usual or normal position of the thyroid in front of the cricoid cartilage and the upper tracheal rings. According to Lahey,[1] the lateral lobes of the thyroid are formed by "projection of cells, like an alveolar or tubulo-alveolar gland." Another anomaly in this descent is the retrosternal or substernal thyroid, which perhaps comes from a projection of cells similar to that which forms the lateral lobes.

As stated, the entire thyroid gland may be on the base of the tongue. I have, however, not been able to find the record of a case in which the entire gland was located in the tongue, in front of the larynx or behind the sternum.

The first authentic case of lingual thyroid was reported in 1888 by Bernays.[2] Dr. M. Lawrence Montgomery,[3] of San Francisco, has made the most extensive study of lingual thyroids. He listed 144 proved cases between 1888 and 1935 and reported his findings at the meeting of the American Association for the Study of Goitre at Salt Lake City in June 1935. He also reported 2 cases of thyroid in the body of the tongue.

Read at the annual meeting of the Spokane Surgical Society, April 30, 1938.

1. Lahey, F. H.: Lingual Thyroid, Surg., Gynec. & Obst. **36**:395, 1923.

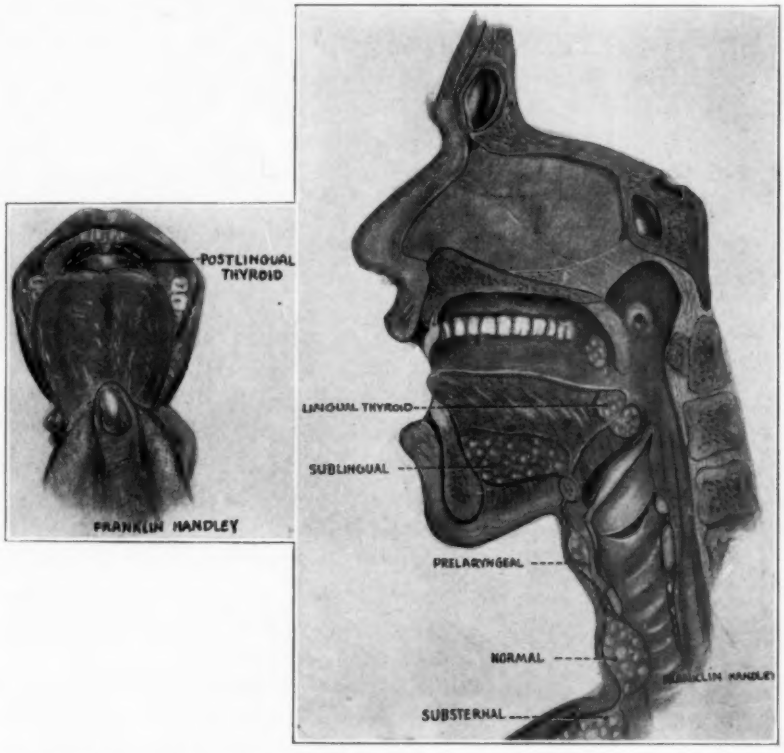
2. Bernays, A. C.: The Origin of the Foramen Cœcum Linguæ, as Shown by an Operation on a Rare Tumor of the Root of the Tongue, St. Louis M. & S. J. **55**:201, 1888.

3. Montgomery, M. L.: Lingual Thyroid, Tr. Am. A. Study Goiter, 1935, p. 145; Lingual Thyroid: A Comprehensive Review, West. J. Surg. **43**:661, 1935; **44**:54, 122, 189, 237, 303, 373 and 442, 1936.

The symptoms of lingual thyroid vary greatly according to the size of the gland, being chiefly due to obstruction and pressure. The gland varies from a few small nodules to the size of a small orange.

In Montgomery's series, 72 patients complained of dysphagia. Inability to swallow was reported in a few cases. Regurgitation of food through the nose occasionally occurs.

Dysphonia was second in frequency, including thickness of voice, nasal tone, change of voice and hoarseness.



Left, postlingual thyroid after operation; the dotted line shows the size of the growth before operation. Right, normal and abnormal positions of the thyroid gland.

Respiratory symptoms came third, the passage of air being much easier than the passage of food, though 1 case of death due to suffocation was reported by Beck. Respiratory symptoms vary from slight difficulty in breathing and attacks of coughing, especially after exertion, to dyspnea.

Hemorrhage occurred in 24 cases, associated with menstruation in 3, and believed to be due to carcinomatous involvement or degeneration in elderly people in 4.

Other symptoms mentioned are tightness or fulness in the throat, pricking sensations in the throat, dizziness, headache and backwardness.

Hypothyroidism was reported in 21 cases in which the thyroid was absent in the neck. Hyperthyroidism occurred in 4 cases of this series.

The age at which symptoms occurred was mentioned in 104 cases. In the males, symptoms occurred during puberty in 36.3 per cent of the cases, and in the females during puberty in 45.1 per cent, and before early maturity, around 30, in 78.5 per cent.

The age of beginning of menstruation and degree of flow was practically the same as in cases of thyroids in the neck. In pregnancy, also, the gland enlarges in the same proportion. The symptoms, however, are more marked simply because of the location of the enlargement.

Malignant growths and degenerative conditions occur in practically the same proportion as in cases in which the thyroids are in normal positions. In cases of simple hypertrophy of lingual thyroids, the glands reduce with iodine therapy in the same proportion as do those affected with simple hypertrophy in the neck.

In cases of thyroid insufficiency, of course, thyroid extract is indicated.

The most frequent route of surgical approach is through the mouth, although in some cases approach is through the neck.

Absence of the thyroid in the neck in practically 70 per cent of these cases warrants an exploratory operation in the neck if there is any doubt of any thyroid being in the normal position before surgical intervention in the tumor on the tongue. If there is no thyroid in the neck, all of that on the tongue should not be removed.

REPORT OF A CASE

About a year ago, during an operation for removal of tonsils and adenoids on a girl of 13, a tumor on the base of the tongue was discovered.

She came under my care in September 1937. At that time she complained of choking sensations and difficult breathing after strenuous exercise or excitement. In play she would frequently have to stop and rest. She never talked well, and her parents said her voice was poorer since the operation; the deterioration was due perhaps to recent hypertrophy, for the tonsils were well removed. She was backward and played less than formerly. Her heart and pulse rate were normal, and she had always been well. Her menstrual periods had not yet started.

On examination, a tumor the size of a walnut was noted on the back of the tongue. This growth covered the entire base of the tongue and was fairly firm, although not firm enough to rule out the possibility of its being a cyst. The growth was anterior to the epiglottis, which was not affected.

With the patient under general anesthesia an aspirating needle was inserted, but no fluid was obtained. A V-shaped incision was then made, and a piece about 1 cm. was removed from the top to the bottom of the growth. There was free bleeding, which was controlled by bringing the edges together with deep catgut

sutures. Microscopic examination showed the specimen to be normal thyroid tissue. A few days later a metabolic test was made, and the rate was found to be + 8 per cent.

Compound solution of iodine U. S. P. was prescribed, 5 minims (0.31 cc.) morning and evening.

The drawing shows that the growth was considerably smaller than before surgical intervention. The dotted line shows the original size. It is between one fourth and one third smaller. The white area along the top of the tumor is the surgical scar.

The patient's voice is now normal. She plays the same as other children and no longer has difficult breathing or choking attacks after exercise. Her backwardness has disappeared, and her general condition is much improved. She has grown 4 inches (10 cm.) since last fall.

Paulsen Medical and Dental Building.

OTITIS MEDIA AND ORBITAL CELLULITIS COMPLICATING SCARLET FEVER

PRELIMINARY REPORT ON LOSS OF HEARING FROM THIS DISEASE

HORACE JAMES WILLIAMS, M.D.
PHILADELPHIA

The material for this paper was obtained from the Philadelphia Hospital for Contagious Diseases during the years 1934 to 1937 inclusive.

The discussion will have reference to the recorded literature on the general subject of otitis media and orbital cellulitis complicating scarlet fever, and a preliminary report on loss of hearing in 104 unselected cases of scarlet fever is included.

OTITIS MEDIA

Scarlet fever produces acute otitis media in more cases than does any other specific disease in this country. A close second place is granted to measles. Some writers would place measles above scarlet fever in their own communities. They may be right. Certainly both are prime producers of aural trouble. I hold the opinion that in Philadelphia the percentage of scarlatinal otitis which proceeds to mastoiditis requiring surgical intervention is substantially higher than that of otitis following measles.

Acute suppurative otitis media is the most important complication of scarlet fever. With the exception of lymphadenitis in general and serum reaction in patients who receive serum, it is also the most frequent complication. In this period of four years, 1934 to 1937 inclusive, 15,140 patients with scarlet fever were cared for at the hospital. In 1,300 of them, or 8.6 per cent, acute suppurative otitis media developed. This percentage may be compared with those given in table 1, which shows the incidence of otitis media in cases of scarlet fever in many epidemics in many parts of the world during a period of many years and under the observation of many different physicians.

It is interesting to note that approximately one third of the patients in whom a running ear developed had bilateral involvement, for among the 1,300 patients with otitis there were 1,792 running ears. In 149, or 8.3 per cent, of these ears, belonging to 115 patients, mastoiditis requir-

Read at the meeting of the American Laryngological, Rhinological and Otological Society, Atlantic City, N. J., April 28, 1938.

ing surgical intervention developed and simple mastoidectomy was performed. The mortality among the 115 patients with mastoiditis was 10, or 8.7 per cent.

The patients with otitis were divided into three groups. In group 1, in which 1,049 ears were involved, the patients were subjected to myringotomy before the tympanic membrane ruptured; in group 2, in which 190 ears were involved, myringotomy was performed after the tympanic membrane had ruptured, and, in group 3, in which 553 ears

TABLE 1.—*Incidence of Otitis Media in Cases of Scarlet Fever*

Authors	Number of Patients with Scarlet Fever	Number of Patients with Otitis Media	Percentage of Otitis Media in Scarlet Fever
Le March'hadour	339	36	10.65
Yearsley	17,829	2,355	13.20
Sprague-Bezold	17,087	64	3.75
Fowler-Bader	51	13	25.00
Biedert	25,882	3,833	14.80
Biedert-Finley	4,397	439	10.00
Biedert-Craiger	1,008	131	13.00
Nager	744	119	16.00
Borden	746	65	8.70
Alderton	1,621	178	11.00
McCrae	850	125	14.70
Ross	2,595	...	14.00
Strachan	589	98	16.60
Lefler	473	58	12.25
Williams	29,298	2,898	9.9

TABLE 2.—*Number of Incisions and Mastoidectomies*

Ears		Number of Incisions					Mastoidectomies			
		1	2	3	4	5	Unilateral	Bilateral	Deaths	Percentage of Mastoidectomies
Incised	1,049	857	144	32	13	3	39	19	6	5.5
Ruptured and incised	190	165	19	6	0	0	17	8	1	13.2
Ruptured	553						25	7	3	5.8
Total	1,792					Total	81	34	10	
Average number of days from operation to recovery										33.47

were involved, the tympanic membrane ruptured and was not incised. The type of myringotomy employed was free linear incision, yet the records show a considerable number of instances in which incision was repeated once or more.

The incidence of mastoiditis requiring surgical intervention was 5.5 per cent in the first group, in which the drum was incised, 13.2 per cent in the second group, in which it ruptured and was incised, and 5.8 per cent in the third group, in which it ruptured and was not incised. In this series of 15,140 patients with 1,792 suppurative ears, early incision of the tympanic membrane did not materially lower the incidence

of mastoiditis requiring surgical intervention. In spite of this interesting point, I advocate early myringotomy; it relieves the pain, tends to reduce the temperature and is the best means of avoiding mastoiditis requiring surgical intervention. There are three factors that still stand out as determining the occurrence of mastoiditis, namely, the virulence of the infection, the resistance of the patient and the individual anatomic structure.

As regards repeated incision of the tympanic membrane, I have come to the conclusion that it is seldom of value, provided that the original incision was free. The condition of the tympanic membrane which invites repeated incision is often due to swollen mucosa of the middle ear irritated by a small amount of pus from the mastoid, which is not sufficient to keep the incision open. If a tympanic membrane has ruptured or been incised and the opening is not sufficient for free drainage, it should be reincised.

TABLE 3.—*Period of Morbidity in Which Otitis Media Occurs*

Week of the Onset of Otitis Media	Ears	Percentage
1	613	34.2
2	502	28
3	257	14.3
4	198	11
5	129	7.2
6	93	5.2
Number of ears	1,792	
Number of patients	1,300	

The period of morbidity in which aural complications occur is of practical interest. Of the 1,792 ears with otitis media, myringotomy was performed on 613 in the first week of morbidity of scarlet fever, on 502 in the second, on 257 in the third, on 198 in the fourth, and 129 in the fifth and on 93 in the sixth.

The data just cited and those given in table 3 support the assertion by Borden that "it may truly be said of scarlet fever that aural complications of any degree of severity may arise at any time from the first day of the acute symptoms to the last day of convalescence." With this opinion I am in complete accord.

Records of the incidence of otitis media according to age in two previous reports showed that approximately 91 per cent of the patients were under 10 years. In the group reported on here 88.6 per cent were under 10 years.

The incidence of scarlet fever is greatest in childhood; 48 to 60 per cent of the patients have not reached the fifth year. The age of the patients probably accounts for the large percentage of tonsils and adenoids present. Of the 1,300 patients with otitis media, 15.7 per cent had had the tonsils and adenoids removed. Of the 14,733 patients

with scarlet fever considered in a report in 1929, 3.5 per cent had had their tonsils removed, and a second report, in 1932, including the data of the first, indicated that 5.8 per cent had had their tonsils removed.

There is a marked seasonal and yearly variation in the incidence of scarlet fever and therefore of scarlatinal otitis. The season in which the incidence is greatest begins about the middle of January and lasts until the middle of May. The virulence of the epidemic varies from year to year, and its association with measles adds much to the severity of the aural disease.

Of the 15,140 patients, streptococcic meningitis developed in 8 and tuberculous meningitis in 2; no abscess of the brain or thrombosis of the lateral sinus developed.

The symptoms of otitis media differ according to whether the complications come early or late. During the initial febrile period they may be accompanied by a further rise in temperature, usually associated with pain and restlessness and more often coming on during the late

TABLE 4.—*Incidence of Otitis Media According to Age*

Age	Number of Patients	Percentage
0- 5	632	48.6
5-10	520	40
10-15	95	7.3
15-20	26	2
20	27	2.1
Total number of patients	1,300	

afternoon or evening, as pointed out by Layton. During the afebrile period there may be a rise in temperature associated with pain and restlessness, but often the disease develops so gradually and asymptomatically that the first sign is a discharge.

In the patients in whom otitis media occurred with the initial symptoms it developed from extension of the infection to the middle ear; in those in whom it occurred later it developed as a result of infection in the sinuses.

Whoople and Cave made roentgenographic examinations of the sinuses of 292 patients with scarlet fever; in 91 per cent of the cases some shadow was seen on the roentgenogram. The sinuses most frequently involved were the antrums and the ethmoid and the frontal sinuses, in the order mentioned. In 11 per cent of the cases otitis media developed. No ear showed purulent otitis media unless there was sinusitis evident by roentgenographic examination on the corresponding side. It is interesting to note that the incidence of involvement of the sinuses was not less in the tonsillectomized patients; it was, in fact, proportionately greater.

The discussion of roentgenographic and laboratory diagnosis is purposely omitted. There has been little change in the treatment during the last twenty years.

Conclusions.—Study of this series of 15,140 patients with scarlet fever leads to the following conclusions:

1. The incidence of otitis media was 8.6 per cent.

2. Between cases in which an early incision of a bulging tympanic membrane was performed and those in which a spontaneous rupture had occurred there was a difference in incidence of 0.3 per cent.

3. Repeated incision of a tympanic membrane is seldom of value.

4. With scarlet fever, aural complications of any degree of severity may arise at any time from the first day of the acute symptoms to the last day of convalescence.

5. The useful principles of treatment available today are essentially those available twenty years ago.

ORBITAL CELLULITIS

Orbital cellulitis is likely to occur repeatedly in the practice of every otolaryngologist, and knowledge of its causation, pathologic process and clinical course is essential to its proper management. Improper conduct may lead to a grave mistake, such as an operation which is untimely or inexpedient or both. The present conception of the causation of orbital cellulitis is comparatively new. Such terms as primary, idiopathic and metastatic in connection with this condition are seldom used, while the terms thrombophlebitic and secondary have become common.

Orbital cellulitis is most frequently due to disease in the paranasal sinuses, and this is true when it occurs as a complication of scarlet fever. Since over 90 per cent of the patients with scarlet fever are children under 10 years of age, it would be expected that infection of the ethmoid sinuses would be the prime producer of orbital cellulitis; such is the case. Infection of the frontal sinus is the most frequent cause in adults. Maxillary sinusitis may produce orbital cellulitis, especially of the lower lid, in young children. As pointed out by Porter and Babbitt, infection from the sinuses may enter the orbit by direct extension with necrosis of the bone, through a dehiscence in the bony wall, along the walls of the vessels or by thrombophlebitis.

There are three stages in the development of orbital cellulitis: first, edema of the lids, frequently confined to the inner half and more marked in the upper lid if the frontal or an ethmoid sinus is involved and in the lower lid if the maxillary sinus is responsible; second, marked edema of the lids, with closure of the palpebral fissure and exophthalmos, usually without limitation of motion; third, evidence of the formation of an

abscess, with or without limitation of motion. There is usually discoloration of the lids, and there may be evidence of pointing, but when the condition is under observation surgical intervention is usually resorted to before pointing appears.

The treatment depends on the degree of involvement. In cases in which there are conjunctivitis, simple edema and cellulitis without the formation of an abscess, it is always conservative. The treatment consists of shrinking the nasal mucosa under the middle turbinate with 1 per cent ephedrine sulfate or cocaine hydrochloride or a combination of the two and aspiration of the nasal cavity by suction produced by a mechanical device attached to a soft rubber catheter. Aspiration is done several times daily, and cold compresses are applied externally.

When the cellulitis continues to increase instead of decreasing after twenty-four to forty-eight hours of conservative treatment, some surgical intervention is necessary. For children whose ethmoid sinuses or antrums are involved, removal of the anterior tip of the middle turbinate and opening of the ethmoid cells, together with intranasal antrotomy when there are symptoms of antral disease, will usually be sufficient for prompt relief of symptoms.

When the edema, discoloration of the lids, exophthalmos and limitation of motion are marked, more radical measures are indicated. Ethmoidectomy by the combined external and intranasal approach is the procedure of choice. In children, the frontal sinuses are usually small and seldom involved enough to require surgical drainage. In the cases of scarlet fever in which surgical intervention was resorted to, only sufficient was done to establish good drainage, extensive surgical procedures being left to be performed after the acute condition had subsided. In this series of cases no extensive surgical intervention was necessary.

Of the 68 patients with orbital involvement, 49, or 72 per cent, were boys. Fourteen, or 20.6 per cent, had had their tonsils and adenoids removed. In 30 the involvement developed in the first week of morbidity, in 21 in the second, in 3 in the third and in 14 in the fourth on the following weeks. In 1 patient it developed on the fifty-eighth day of disease.

In this respect it resembles otitis media complicating scarlet fever, viz., occurring at any time from the first day of the disease to the last day of convalescence. Each patient had one or more sinuses involved. The number of days from onset to recovery varied from forty-eight hours to forty-five days.

In this series of 68 cases the pathologic process consisted of conjunctivitis, edema, cellular infiltration without suppuration and periorbital abscess.

There were 10 cases of marked conjunctivitis without any other orbital involvement; yet in many of the other cases the condition began with conjunctivitis or the latter was present during the course of the orbital cellulitis. The average duration of the conjunctivitis in the 10 cases was four days.

Simple orbital edema without any evidence of inflammation involving the inner half of the upper lid occurred in 18 patients. Forty-two had advanced orbital cellulitis, varying from moderate swelling to complete closure of the palpebral fissure. The average length of time from beginning to subsidence of the inflammation in the cases in which the records were complete and it could be computed was five days.

Ten patients received surgical treatment. Two patients, aged 10 and 7 respectively, had ethmoiditis and frontal sinusitis. A combined external and internal operation was performed. A perforation in the floor of the frontal sinus was present in both cases and thought large enough for drainage. Three patients had a combined external and

TABLE 5.—*Period of Morbidity in Which Orbital Cellulitis Occurs*

Week of the Onset of Orbital Cellulitis	Patients
1	30
2	21
3	3
4	14
Number of patients	68

internal operation for orbital abscess as a result of ethmoiditis. In each of these cases a perforated tube was placed in the wound, extending to the nose for drainage. Two of the patients had maxillary sinusitis, which was irrigated.

There were 5 for whom the abscess only was opened in combination with local nasal treatment. Twenty-two of the 68 patients had otitis media, and mastoiditis developed in 6. Four of the 68 died, 1 of meningitis of undetermined origin, 1 of purpura haemorrhagica, 1 of pulmonary abscess and 1 of abscess of the thigh.

PRELIMINARY REPORT ON LOSS OF HEARING FROM SCARLET FEVER

One hundred and four patients responded to the request which was sent from the hospital to persons who had had scarlet fever to return for an examination of their hearing. No subject under the age of 10 years was examined. Each subject had had scarlet fever one to four years prior to examination.

The examinations were conducted principally at the Municipal Hospital for Contagious Diseases, in a small room between the office and the library. The examinations were made on Saturday afternoons,

when the adjacent rooms were not in use and noise was at a minimum. A 2-A Western Electric audiometer was used.

The subjects were divided into four classes:

1. Those who had had no known involvement of the ear.
2. Those who had had earache but no discharge.
3. Those who had had discharging ears.
4. Those who had had mastoidectomies.

Fifty-six subjects had had no known involvement of the ear. Twenty-five of these had had tonsils and adenoids removed previous to the onset of scarlet fever; the average loss of hearing was 7.9 per cent. In 31 cases tonsils had not been removed; the average loss of hearing was 6.7 per cent. Twelve gave a history of some form of sinusitis; the average loss of hearing was 7.5 per cent. In this group there were 3 with a moderate loss of hearing for high tones.

Fifteen subjects had had earache during scarlet fever but had had no discharge. Eleven of these had had tonsils and adenoids removed previous to the onset of scarlet fever; the average loss of hearing was 5 per cent. In 4 cases tonsils had not been removed; the average loss of hearing was 9.2 per cent. Six persons gave a history of some form of sinusitis; the average loss of hearing was 5.6 per cent. These subjects showed no loss for high tones.

Twenty-four subjects had had discharging ears during scarlet fever. Fifteen of these had had tonsils and adenoids removed previous to the onset of scarlet fever; the average loss of hearing was 10.5 per cent. In 9 cases tonsils had not been removed; the average loss of hearing was 10.2 per cent. Seven subjects gave a history of some form of sinusitis; the average loss of hearing was 14 per cent. Four of these had a moderate loss for high tones.

Nine subjects had had mastoidectomies during scarlet fever. One gave a history of sinusitis; the tonsils had been removed previous to the onset of scarlet fever; the average loss of hearing was 9.9 per cent.

The outstanding observations in these examinations were:

1. The loss of hearing was greatest in subjects who had had discharging ears.
2. Removal of tonsils had little effect on loss of hearing.
3. The history of sinusitis had but little effect except when it was associated with otitis media.

I propose to extend the study to a larger number and report the findings in a later paper.

THE LARYNX IN INFANCY

A STUDY OF CHRONIC STRIDOR

CLYDE A. HEATLY, M.D.
ROCHESTER, N. Y.

The larynx in infancy presents certain conditions of acute or chronic nature which differ sufficiently from those encountered in later life to be of great interest and importance. The perfection of the technic of direct laryngoscopic examination has naturally removed the uncertainty which previously obscured the diagnosis and management of these disorders. It is the more surprising therefore to note the frequency with which this important procedure is still deferred or altogether omitted. It should be considered axiomatic that any stridor observed in infancy requires careful direct laryngoscopic examination regardless of other clinical findings which may apparently explain the disturbance. This contention is repeatedly emphasized in the study of 20 cases of chronic stridor in infancy on which this thesis is based.

EMBRYOLOGIC CONSIDERATIONS

A brief résumé of the more important phases of the development of the larynx is indispensable to an understanding of certain abnormalities encountered in infancy. It is an interesting clinical fact that despite the complicated formation true congenital malformations are exceedingly rare.

The larynx, according to Frazer,[1] is a compound formation consisting of an infraglottic portion, formed around the upper end of the tracheal tube, and a supraglottic portion, formed by a modification of the pharyngeal floor lying immediately around the sagittal, slitlike orifice of the pulmonary outgrowth (primitive glottis). The third, fourth and sixth branchial arches are intimately concerned with its development. By the fifth week of fetal life the two sixth arches have enlarged, forming on each side of the primitive glottis and behind the hypobranchial eminence two rounded masses identified as the arytenoid

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From the Division of Oto-Laryngology, University of Rochester School of Medicine and Dentistry.

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1. Frazer, J. E.: The Development of the Larynx, J. Anat. & Physiol. **44**: 156, 1909-1910.

swellings. During the seventh week these rapidly growing swellings fold themselves upward and forward, thus adding a transversely disposed cavity to the original sagittal slit and forming a T-shaped glottis behind the eminence. At this time the arytenoid swellings fuse and for a short period obliterate the primitive glottis, presumably in order to prevent the flow of amniotic fluid into the pulmonary outgrowth. This fusion dissolves during the tenth and eleventh weeks. The ventricle of the larynx begins in the latter half of the second month as an outpouching of the lateral wall of the transverse cavity. As the arytenoid fusion disappears, the ventricle increases in size, and, as a result, superior and inferior bounding folds are formed. The former becomes the ventricular band and the latter the true vocal cord, bounding the definite glottis. The epiglottis is directly but gradually derived from the hypobranchial eminence under the molding influence of the third arch. The remains of the third arch connecting it to the sides of the pharynx become the pharyngoepiglottic folds. The fourth arch also is connected with it and becomes the aryepiglottic fold.

ANATOMY

The larynx is fully developed at birth and increases slowly in size until puberty. In infancy there is no difference in size according to sex. The original position is high, at the level of the fourth cervical vertebra, but with growth it slowly descends to reach the sixth or seventh cervical vertebra in adult life. As the result of its original high position, its entrance is at a sharp angle from behind, forward and downward toward the glottic lumen. The structures bounding the upper aperture of the infantile larynx are soft and readily collapsible, as was so ably demonstrated by the early experiments of Thomson and Turner.[2] One important factor in this is that the epiglottis, the cartilages of Wrisberg and the apexes of the arytenoid cartilages are made up of pliable, yielding elastic cartilage in contradistinction to the remainder of the larynx, which consists for the most part of brittle hyaline cartilage. The infantile epiglottis is narrow and folded longitudinally, presenting a deep gutter-like posterior aspect. In consequence the soft yielding aryepiglottic folds are drawn together so as to effect a relative narrowing of the upper laryngeal aperture.

The infantile glottis is also proportionally smaller. According to Tucker[3] the anteroposterior measurements vary from 7 to 9 mm., with the subglottic larynx 1 or 2 mm. smaller in diameter in the same subject.

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2. Thomson, J., and Turner, A. L.: On the Causation of the Congenital Stridor of Infants, Brit. M. J. **2**:1561,-1900.

3. Tucker, G.: The Infant Larynx: Direct Laryngoscopic Observations, J. A. M. A. **99**:1899 (Dec. 3) 1932.

The latter measurement is of great importance, because the subglottic area is completely surrounded by the cricoid ring and therefore is not capable of instrumental dilation. Tucker's measurements for a series of 30 infants up to 2 months of age indicate that a subglottic measurement of 6 to 8 mm. may be considered normal for a well developed infant. A diameter of 4 mm., however, constitutes a true and usually dangerous stenosis. The cartilages of the larynx of the infant are much softer than those of the adult and therefore more liable to collapse from negative pressure in the case of a laryngeal condition associated with stenosis. The mucous membranes are more vascular, as well as more loosely attached, in infancy. The reflex irritability also is far greater. These factors are responsible for the easily provoked edema and spasm so frequently encountered as the result of acute inflammation or instrumental manipulation in the larynx during infancy.

TYPES OF STRIDOR

As a basis for this study of chronic stridor in infancy a series of 20 cases encountered during the past eleven years was reviewed. The method of investigation pursued in each case was the same. After a complete physical examination, fluoroscopic and roentgen studies of the neck and mediastinum were carried out. Direct laryngoscopic examination was then performed without anesthesia. A summary of the final diagnosis in each case is given in the following tabulation:

Final Diagnosis	Number of Cases
Congenital laryngeal stridor	12
Paralysis of the vocal cord	2
Tumor of the larynx	1
Congenital web	1
Subglottic tumor	1
Subglottic stenosis	1
Tumor of the mediastinum	1
Congenital anomaly of the ligamentous attachments of the sternum (?)	1
Total	20

Congenital Laryngeal Stridor.—It is generally agreed that this diagnosis should be limited to cases in which there is an exaggerated infantile type of larynx. While the history in itself is frequently suggestive, it must be emphasized that the tendency to apply this diagnosis without the complete examination already outlined is deplorably common. Congenital laryngeal stridor is usually noticed at birth or shortly after. It is typically inspiratory, although in severe attacks an expiratory croak is usually heard. The stridor is aggravated by crying and tends to diminish or disappear during sleep or nursing. It is of importance to emphasize

that the vocal sounds are unchanged and that actual cyanosis is infrequently observed. Retraction in the epigastrium and the suprasternal notch is apparent in the more severe attacks. The stridor characteristically disappears during the second year, with the normal growth of the larynx. Fatalities, however, have been reported as the result of intercurrent infection of the respiratory tract. Blackader and Muckleston [4] found that in one sixth of the cases reported the patient died of infection of the respiratory tract.

A great mass of literature on this subject has accumulated since the first case was reported by Lees in 1883.[5] The early records may be dismissed in many instances because of the lack of facilities for direct laryngoscopic examination. The first careful study was reported in 1900 by Thomson and Turner.[2] They concluded that the stridor was caused by poor coordination of the respiratory muscles, resulting in the typical narrowing of the upper laryngeal orifice. The fact, however, that the stridor is noticed at birth or shortly after is decidedly against the validity of this assumption.

Direct examination reveals an exaggeration of the characteristics of the normal infantile larynx. The epiglottis is elongated and sharply folded on itself. In some instances it is unusually broad and flaccid. The aryepiglottic folds are flabby and apparently unsupported. The arytenoid cartilages may appear long and tapering. The stridor is clearly caused by the vibrations of the elongated epiglottis and the flabby structures of the upper rim of the larynx as they are sucked in toward the glottis during inspiration. Hill [6] has clearly summarized the most plausible etiologic factors in this condition: "There must be some local condition, some variation or congenital malformation in the structures themselves of this portion of the larynx which is the essential factor in at least a majority of these cases."

While the foregoing description fits the condition usually encountered, attention should be directed to the reports of certain interesting variations. Hasslinger [7] reported 3 cases in which there was an edematous ridge in the mucous membrane of one or both aryepiglottic folds, which obstructed the glottis during inspiration, causing stridor. In 2 of these cases the ridge was removed, with immediate relief of symptoms. Microscopic examination showed spongy edematous connective tissue with few cells and covered with pavement epithelium.

4. Blackader, A. D., and Muckleston, A. S.: Inspiratory Stridor and Dyspnea in Infants, Arch. Pediat. **26**:401, 1909.

5. Lees, D. B.: Larynx from an Infant with a Peculiar Form of Obstructed Respiration, Tr. Path. Soc. London **34**:19, 1883.

6. Hill, F. T.: Congenital Laryngeal Stridor, Laryngoscope **40**:44, 1930.

7. Hasslinger, F.: Zur Pathogenese, Diagnostik und Therapie des Stridor congenitus, Ztschr. f. Hals-, Nasen- u. Ohrenh. **21**:222, 1928.

Thomas [8] reported the postmortem observations in 2 cases in which the larynx was normal but there was well marked evidence of intracranial hemorrhage resulting from injury at birth. He concluded that congenital stridor may occasionally be of central origin without laryngeal deformity. Putzig [9] and Tucker [8] have each reported a case in which stridor was primarily caused by an abnormally small larynx. Iglauer [10] has recorded a most unusual case in which actual epiglottidectomy was necessary to relieve frequently recurring attacks of asphyxia. The stridor gradually cleared after about twelve days, and no disturbance in swallowing was observed. Histologic examination of the epiglottis gave entirely normal results. Rooker [11] reported a similar case, in which amputation of the epiglottis was successfully performed. Hill [6] described the unusual case of a 2 week old infant, in which a twisting motion of a flabby omega-shaped epiglottis was observed. On inspiration the right margin rolled downward and inward followed by the entire epiglottis, which was sucked down and then expelled with a croaking sound. This child's condition gradually cleared without treatment.

The history and findings in 2 cases in my own series appear of sufficient interest to be described briefly.

C. T., a 1 year old girl, was first seen on May 10, 1933, with a history of coarse, stridorous breathing and recurring attacks of severe dyspnea since birth. She was poorly developed and badly nourished. There was a loud inspiratory stridor, accompanied by well marked abdominal and thoracic retraction without cyanosis. The lungs were entirely clear. Roentgen and fluoroscopic examination showed entirely normal conditions. The principal finding by direct laryngoscopic examination was an unusually broad and flaccid epiglottis, which on inspiration was drawn downward, almost completely covering the glottis. Although a long tapering epiglottis was noted in a number of the other cases, in no other did it approach the development seen in this case. In retrospect, it seems decidedly of the type described by Iglauer and Rooker. No surgical intervention, however, was permitted. The parents were instructed to watch the child carefully, especially in the event of an acute infection of the upper part of the respiratory tract. The infant was examined in the pediatric department two years later and reported relatively free from stridor except during marked excitement. There was no opportunity for subsequent laryngoscopic examination.

M. R., a poorly nourished boy of 3 months, was examined on Oct. 21, 1936. There was a history of well marked inspiratory stridor since birth. At the age of 3 weeks this child had been seriously ill for two weeks with ulcerative

8. Thomas, E.: Zur Klinik des Stridor congenitus, Monatschr. f. Kinderh. **22**:223, 1921.

9. Putzig, H.: Zur differential Diagnose von Thymus Hypertrophie und kongenital Stridor, Ztschr. f. Kinderh. **35**:322, 1923.

10. Iglauer, S.: Epiglottidectomy for the Relief of Congenital Laryngeal Stridor, with Report of a Case, Laryngoscope **32**:56, 1922.

11. Rooker, cited by Iglauer, in discussion on New.[18]

stomatitis, during which time the respiratory symptoms had been greatly aggravated. The clinical and roentgenologic studies showed no cause for the stridor. On direct laryngoscopic examination an unusually broad, elongated, flabby epiglottis was discovered. It was noted that on inspiration the left margin rolled downward and under, and this movement was followed by a drawing downward of the remainder of the epiglottis. The left cord appeared thickened and slightly reddened, probably as a result of the severe infection of the mouth two months before. This child has been admitted to the hospital twice subsequently for bronchopneumonia, but his condition has improved so greatly that, according to the attending pediatrician, the stridor is now noticed primarily during excitement. This case presents an interesting similarity to that described by Hill, in which also the condition gradually cleared without treatment.

Paralysis of the Vocal Cords.—Paralysis of one or both vocal cords as a cause of chronic stridor in infancy has been not infrequently observed. Associated evidence of laryngeal obstruction also has been noted in the presence of bilateral palsy, and, as Jackson [12] has emphasized, in many cases asphyxia neonatorum from this cause has passed unrecognized, with fatal termination. The recurrent palsy is usually the result of prolonged difficult labor with injury to the nerves from traction or instrumental pressure. Similar trauma may result also in injury to the cricoarytenoid joints, with fixation or dislocation of the arytenoid cartilages. Jackson [12] has reported examples of this type of injury with gradual disappearance of the resulting stridor. In most instances the palsied cord assumes the midline position. Jackson,[12] however, has described 1 case in which a newborn infant with the cords in the cadaveric position gradually became normal in two weeks. New [13] reported a most interesting instance of bilateral abductor palsy, with midline position, in an infant of 10 months. There was a history of dyspnea since birth. No treatment was given, and subsequent reports indicated gradual clearing. This is the course usually observed in such cases. Occasionally the stridor is sufficient to necessitate tracheotomy; even then, however, the paralysis gradually disappears, which permits successful decannulation. Myerson [14] described a case of complete palsy in a newborn infant with fatal termination in spite of prompt tracheotomy. At autopsy an extensive epidural and subdural hematoma, mostly in the posterior fossa, was discovered. These experiences seem to emphasize the possibility of instrumental damage to the recurrent nerves or laryngeal structures during delivery, with resulting disturbances in the form of stridor, dyspnea or actual asphyxia. Only prompt

12. Jackson, C., and Jackson, C. L.: The Larynx and Its Diseases, Philadelphia, W. B. Saunders Company, 1937, pp. 64-74.

13. New, G. B.: Congenital Obstruction of the Larynx and Pharynx, J. A. M. A. **81**:363 (Aug. 4) 1923.

14. Myerson, M. C., in discussion on Clerf, L. H.: Congenital Stenosis of the Liver, Tr. Am. Laryng. A. **53**:207, 1931.

laryngoscopic examination will show the cause and indicate the proper method of management.

Two cases of abductor palsy were encountered in this series.

C. H., a girl of 4 months, was brought in with several complaints, including chronic stridor since birth. Birth had been premature, at 7½ months, after a difficult, prolonged labor. At birth a spina bifida, with meningocele had been noted. This had increased considerably in size. Evidence of internal hydrocephalus also was present. Both legs had been paralyzed since birth. Fluoroscopic and roentgen studies of the neck and chest gave negative results. Direct laryngoscopic examination showed bilateral abductor palsy, with both cords in the median position. The condition was considered hopeless by the pediatric and neurologic consultants, and the patient was discharged untreated.

B. B., a newborn infant, was seen on March 2, 1927, with vomiting, noisy, labored respirations and intermittent cyanosis. Clinical and roentgenologic studies of the chest gave negative results. Direct laryngoscopic examination showed bilateral abductor palsy, with both cords in the median position. A few hours later severe nasal bleeding began. A lumbar puncture was done and blood noted in the spinal fluid. The child died two days later. The final diagnosis was hemorrhagic disease of the newborn, early and severe.

Tumors of the Larynx.—Obstruction of the infant larynx due to any form of neoplasm is relatively unusual. The commonest tumor encountered is the papilloma, which is characteristically multiple and, according to Jackson,[12] may actually be congenital. The youngest patient reported in his series was a newborn infant. In New's[15] series of 40 cases reviewed in 1921, the youngest patient was 10 months old. Angioma also is occasionally encountered in infancy. This tumor may be primary in the larynx but more commonly is associated with similar tumors about the face or the upper air passages. The youngest patient on record was a 10 week old infant, whose case was reported by Levbarg.[16] New and Clark[17] reported the case of an infant of 9 months who presented the symptoms of rattling dyspnea and inspiratory stridor. Radium was used successfully and was advised as the treatment of choice. Cyst of the larynx in infancy is rare. Jacobi and Rascoff[18] in presenting 2 cases in 1935 stated that only 11 cases had previously been reported. They found that cyst involving the epiglottis in the newborn is still more unusual, discovering only 1 reported case previous to their own. The earliest resulting disturbance, labored stridorous breathing, is usually observed at birth and is in most instances con-

15. New, G. B.: Treatment of Multiple Papillomas of the Larynx in Children, Ann. Otol., Rhin. & Laryng. **30**:631 (Sept.) 1921.

16. Levbarg, J. J.: Intubation for Angioma of the Larynx in a Child Ten Weeks Old, Laryngoscope **28**:867, 1918.

17. New, G. B., and Clark, C. M.: Angiomas of the Larynx: Report of Three Cases, Ann. Otol., Rhin. & Laryng. **28**:1025 (Dec.) 1919.

18. Jacobi, M., and Rascoff, H.: Cyst of the Larynx in Infancy, Am. J. Dis. Child. **49**:448 (Feb.) 1935.

tinuous. Flexion of the head usually results in a prompt increase in the respiratory distress, in contrast to the relative improvement which follows turning of the head backward or sideward. This, according to Jacobi and Rascoff, is an important observation in differential diagnosis. While the majority of patients in reported cases died as the result of obstruction, 4 survived as the result of incision and drainage (Park and Israel,[19] Lorie and Lux [20] and Chamberlain [21]) or spontaneous rupture (Horne [22]).

Only 1 case of tumor was encountered in this series. Multiple papilloma was observed in an infant of 13 months; hoarseness had begun six months before. Repeated removals by the scalping technic of Jackson, with early tracheotomy, finally resulted in a cure after two years.

Congenital Web.—Congenital web of the larynx is of infrequent occurrence in the experience of most laryngologists, although Jackson [12] has encountered 84 cases. It is usually found as a membrane uniting the vocal cords at the anterior commissure and extending backward for a variable distance to a crescentic margin. Occasionally subglottic or supraglottic web has been reported. Structurally the web usually consists of a wedge-shaped mass of tissue, thickest at the anterior commissure, and may contain connective tissue, muscle fibers, rudimentary glands and occasionally cartilage. It must be carefully differentiated from an acquired diaphragm resulting from injury or ulceration of the laryngeal mucosa. The acquired type may involve any portion of the larynx or subglottic space. The congenital web is caused by an arrested separation of the fusion which normally obliterates the primitive glottis during the middle of the second month of fetal life. Under normal circumstances, as has already been mentioned, this epithelial fusion completely dissolves during the third month, the dissolution proceeding from behind forward, reestablishing the glottis.

The symptoms resulting from a congenital web depend on the degree of stenosis. A small web may be entirely symptomless and only be discovered later in life during routine examination. A larger one may cause stridor and serious interference with respiration and phonation from birth.

Universal experience in the treatment of congenital web indicates that simple incision or excision is promptly followed by recurrence, with troublesome adhesions. For that reason a small web causing no symp-

19. Park, J. H., and Israel, S.: Epiglottic Cysts in Infancy, South. M. J. **18**:128, 1925.

20. Lorie, A. J., and Lux, P.: Congenital Cyst of the Larynx, Laryngoscope **42**:957, 1932.

21. Chamberlain, W. B.: Report of a Case of Cysts of the Larynx, Laryngoscope **27**:622, 1917.

22. Horne, J.: Cyst of the Epiglottis, J. Laryng. **13**:57, 1898.

toms is best left alone. Repeated incisions and bougienage are necessary to prevent union of the raw edges. Jackson [12] advocated an incision to release the web on one side only, followed by daily endoscopic dilations to prevent union. Later, if necessary, the redundancy on the other side may be removed. Surgical diathermy has been advised by Arbuckle [23] and others as a means of destroying the scar tissue. More recently Iglauer [24] has suggested a two stage operation utilizing the experience gained by general surgeons in the treatment of syndactylism. The procedure consists of "inserting an indwelling spring ring through a puncture in the web at the anterior commissure. After epithelization has occurred about the ring, the web is removed by a V-shaped excision with its apex at the newly formed commissure." The epithelium-lined commissure prevents adhesions from reforming.

One case of congenital web was encountered in this series. In a 13 month old infant with persistent stridor and evidence of moderate laryngeal obstruction since birth, the web united the anterior two thirds of both vocal cords, ending in a crescentic margin. The posterior portion was thin and easily broken through with metal dilators. In the region of the anterior commissure, however, the web was thick and required removal with triangular punch forceps. Obstructive edema complicated this procedure and necessitated temporary tracheotomy. After numerous dilations the edges of the vocal cords healed, and the larynx remained clear except for a slight residual thickening in the anterior commissure.

Subglottic Stenosis.—Chronic stridor in infancy may occasionally result from an obstruction in the subglottic area. A congenital subglottic web is rarely encountered, because interference with the dissolving of the temporary fetal fusion is far less likely to occur in this region than in the relatively narrow area of the primitive glottis. New [13] reported a case of subglottic diaphragm in a child of 1 year with a history of stridor and asthmatic breathing at intervals since birth. An emergency tracheotomy had been performed three and one-half months before his examination. The tube was successfully removed one year later without direct treatment to the subglottic obstruction. It is of interest to note that the thymus had erroneously been considered the cause of the respiratory disturbance and radium had been used on the neck without benefit.

Two of the most interesting cases in this series were of abnormalities in the subglottic area and will be reported in detail.

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23. Arbuckle, M.: Treatment of Cicatricial Stenosis of the Larynx, Tr. Am. Laryng. A. **54**:63, 1932.

24. Iglauer, S.: A New Procedure for the Treatment of Web in the Larynx, Arch. Otolaryng. **22**:597 (Nov.) 1935.

R. C., a 2½ month old boy, was admitted to the hospital with a diagnosis of probable thymic enlargement. Stridor was noted shortly after birth, and there had been periods of actual dyspnea. About two weeks before admission breathing became increasingly labored, and for the preceding twenty-four hours the dyspnea had become alarming, with periods of marked cyanosis.

Examination showed a fairly well developed infant in marked respiratory distress, with both inspiratory and expiratory stridor. The chest was clear clinically, and neither fluoroscopic nor roentgen study showed any abnormality in the chest, neck or mediastinum. The child was placed in a croup tent, but the evident progression of signs of obstruction necessitated tracheotomy a few hours later. This promptly relieved the dyspnea. After a few days of rest, direct laryngoscopic examination was performed. The epiglottis and laryngeal structures were normal. About 1 cm. below the left vocal cord, however, a large mass was discovered protruding from the posterior wall of the subglottic tissues. The mass was firm on probing and was large enough to prevent the passage of a 3 mm. bronchoscope. It was removed by biting forceps, the work being done through a 5 mm. tracheoscope in order to separate and protect the vocal cords. A second inspection five days later showed that a small remnant persisted, and this was satisfactorily removed. Decannulation was possible ten days later, and the child was discharged from the hospital shortly afterward. No subsequent respiratory difficulty has been noted. The biopsy report was as follows: "Section reveals a polyp-like structure formed of dense fibrous tissue covered with pseudostratified columnar epithelium and containing many small glands."

J. F., a 3 month old girl, was seen in consultation because of stridor and alarming chronic respiratory obstruction of undetermined origin. The preceding history, as summarized from the descriptive notes of the attending pediatrician, is unusual enough to report in some detail. The child was born at full term and delivered by forceps. Shortly after birth a small hemangioma was noticed at the right corner of the mouth. At the age of 7 weeks the child was seen by the attending pediatrician because of stridor and marked obstructive dyspnea. There was no evidence suggesting an underlying acute infection. It was noted that the voice was normal. A definite widening of the area of dulness over the upper sternum was detected clinically and confirmed by fluoroscopic and by roentgen examination. In the latter the trachea appeared deviated to the right and partially compressed.

The impression was that symptoms were caused by an intrathoracic mass, probably an enlarged thymus. A roentgen treatment was promptly given because of a sudden increase in dyspnea during the examination. Oxygen was administered by means of a nasal funnel. The following morning the child's condition was unimproved. There were rare moments when she seemed to breathe with ease, but for the most part dyspnea was intense, with marked retraction of the soft parts of the thorax, opisthotonos and slight cyanosis. Two laryngologists in consultation advised against direct laryngoscopic examination or tracheotomy in view of the critical condition of the infant and the apparently established evidence of intrathoracic tumor. The child was transferred to a hospital where an oxygen tent was available. Another roentgen treatment was given. The following day the child seemed definitely better and was removed from the oxygen tent. Two days later she was discharged from the hospital.

Two weeks later, at the age of 2½ months, she was again admitted to the hospital because of dyspnea. In the interval she had had one roentgen treatment over the thymus gland and until a few days before had done well. It was now

thought that she had an intrathoracic tumor which was not simple hypertrophy of the thymus. Because of the progression of the hemangioma on the child's face, it was considered that the tumor might be an angioma, despite the fact that this is one of the rarest of intrathoracic tumors. It was thought that this type of tumor might explain the occasional remissions of dyspnea. As the outlook appeared hopeless unless the dyspnea could be relieved, it was decided to explore the mediastinum. This was done with the patient under ether anesthesia. The sternum was split from the jugular notch to the xiphoid process. Ether and oxygen with positive pressure were then administered. A mass presenting the appearance of normal thymus gland was removed. No other tumor could be detected. The weight of the mass removed was 12 Gm. Microscopically it appeared to be normal thymus tissue, with surprisingly little fibrosis in spite of the previous roentgen therapy. The child withstood the operation well and was returned to the oxygen tent breathing quietly. Thirteen days later she was discharged from the hospital. At this time moderate dyspnea was observed but was attributed by the surgeons to the loss of a fixed thoracic point temporarily resulting from the operation.

Four days later the child was readmitted to the hospital in severe respiratory distress. The thought then occurred that the site of the obstruction might be localized by the injection of iodized poppyseed oil through the laryngoscope. I was called in for this study. The subglottic tissues were seen to be markedly swollen, but the child's condition was too critical to permit a more thorough study. Roentgenograms were taken after the injection of 5 cc. of warmed iodized poppyseed oil into the trachea. These showed a normal tree, with no evidence of extrinsic compression. It was therefore decided that the origin of the respiratory obstruction was not in the thoracic portion of the trachea, as previously considered established, but actually in the larynx itself. A tracheotomy was therefore performed, and the dyspnea promptly ceased. A few days later a careful direct laryngoscopic study was possible. The epiglottis, ventricular bands and vocal cords appeared normal. The subglottic space, however, presented a most interesting picture. A thick band of somewhat reddened tissue projected beneath each vocal cord, reducing the lumen to a narrow slit. Probing demonstrated this tissue to be rather soft and readily displaced but distinctly not edematous. It presented none of the characteristics of a hemangioma. In my opinion it represented an unusual redundancy of the subglottic tissue. After the infant recovered from the effects of the tracheotomy, she was subjected to periodic dilations of the subglottic obstruction; a no. 12 metal bougie was used first and the size gradually increased to no. 24. Nine treatments were given over a period of sixteen weeks, and progressive improvement was apparent. At the end of the fifth week, the child could breathe fairly well with the tracheotomy tube occluded, and after thirteen weeks the tube was removed. No further respiratory difficulty occurred. When the child was last examined at the age of 9 months, breathing was normal; the chronic stridor had completely disappeared, and the subglottic bands were seen to have retracted to the edges of the vocal cords.

So far as my associates and I [25] have been able to discover, this type of congenital deformity, which may be described as congenital hyperplastic redundancy of the subglottic tissues, has not been previously reported. It is of interest to note, however, that Jackson [12] has reported

25. Glaser, J.; Landau, D. B., and Heatly, C. A.: Subglottic Laryngeal Stenosis in Infancy, Am. J. Dis. Child. **50**:1203 (Nov.) 1935.

2 cases of chronic stridor in infancy caused by hypertrophy of the normally inconspicuous ventricular bands, as well as 1 case of myxedematous stenosis in congenital cretinism.

EXTRINSIC CAUSES

The most important extrinsic cause of chronic stridor in infancy is tumor of the neck or superior mediastinum encroaching on the larynx or upper trachea. The thymus may occasionally be the site of a benign tumor, such as a cyst, lymphangioma, lipoma or congenital myxoma, but in nearly all cases the primary thymic tumor has been malignant (thymoma). Jackson [12] has reported 1 case of anteroposterior tracheal compression by congenital intrathoracic goiter in which it was necessary to perform tracheotomy and maintain a long cane-shaped tube in place for six months. He has also described 6 unusual cases of so-called pseudosyrinx, characterized by symmetric mounding inward of the lateral tracheal walls, producing a sagittal slitlike lumen, with resulting stridor. No treatment was required for any of these infants. The condition is apparently a congenital malformation and not caused by external compression. Both New [18] and Fetterolf [26] have reported cases of respiratory disturbance in infancy from a lingual thyroid. New [18] has also described a most unusual instance of the successful management of respiratory distress caused by congenital flaccidity of the tongue and palate.

Chronic stridor in infancy is frequently erroneously considered of thymic origin because of an enlarged shadow reported in the fluoroscopic or roentgen examination without direct inspection to exclude a laryngeal cause. In 11 cases of this series such a diagnosis had previously been made, and only the failure of roentgen therapy to influence the symptoms was responsible for further study. In the case just described, an enlarged thymus had actually been removed before the real cause of the respiratory distress was discovered in the larynx itself. In a series of 15 cases of chronic stridor reported by Kennedy and New [27] in 1931, a diagnosis of enlarged thymus had previously been made in 9, and in all but 1 roentgen therapy had been administered without benefit. In 13 of these cases it was possible to make a final diagnosis, and in only 1 instance was a tumor of the thymus established. During the ten year period covered by this study no case was observed in which symptoms were directly referable to simple hypertrophy of the thymus. It is an important clinical fact that in the hands of many roentgenologists the roentgenogram does not give a true picture of the

26. Fetterolf, G.: A Case of Lingual Thyroid, Laryngoscope **22**:1015, 1912.

27. Kennedy, R. L. J., and New, G. B.: Chronic Stridor in Childhood, J. A. M. A. **96**:1286 (April 18) 1931.

size of the thymus gland. Pancoast,[28] recognizing this, has emphasized the importance of the lateral views in both the inspiratory and the expiratory phase. Hasley and DeTomasi,[29] by cinex camera studies, have demonstrated that the size of the thymic shadow varies not only with the phase of respiration but with the phase of the cardiac cycle. Careful fluoroscopic studies from both the anteroposterior and the lateral position are necessary to the recognition of these variations. It seems imperative in the presence of persistent stridor or dyspnea in infancy that the final diagnosis be withheld until careful direct inspection of the larynx and tracheobronchial tree has been completed. Such an examination is particularly indicated whenever roentgen treatment of an apparent enlargement in the superior mediastinum has not resulted in prompt relief of symptoms.

One case of mediastinal tumor was observed in this series.

J. F., a 3 month old girl, was brought in on Nov. 16, 1934, because of noisy breathing since birth. By the age of 5 weeks, the stridor had markedly increased, and actual signs of obstructive dyspnea were observed. At this time a roentgenogram of the chest was made and a wide shadow reported in the superior mediastinum, presumably an enlarged thymus. Two roentgen treatments were given without relief. Because of the increasing respiratory distress, the child was brought into the hospital for study. Examination at this time showed a poorly nourished infant, appearing chronically ill. There was marked inspiratory and expiratory stridor but no cyanosis. The chest was poorly formed, with flaring of the costal margins, a well marked Harrison's groove and beading of the costochondral junctions. Well marked retraction was noted in the soft spaces about the thorax. The superior mediastinal dulness was definitely widened to percussion. The breath sounds were obscured by the stridor, but a few moist rales were reported at both bases. The roentgenogram showed a broad shadow in the superior mediastinum extending well over into the left lung. At bronchoscopic examination the larynx was found to be normal. The trachea was markedly deviated to the right and compressed in the region of the bifurcation. The consensus was that the condition was a mediastinal tumor, and irradiation was advised. This child died two months later, but no autopsy was permitted.

The following case is of considerable interest because of the diagnostic difficulties presented by the possibility of accompanying tumor.

B. R., an infant of 3 months, was admitted on Oct. 12, 1936, because of noisy respirations since birth. The child had been examined by numerous physicians and had received roentgen treatments for a supposedly enlarged thymus without benefit. Examination showed a well nourished infant with marked inspiratory stridor. Even on quiet breathing, considerable retraction was noted in the epi-

28. Pancoast, H. K.: Roentgenology of the Upper Respiratory Tract with Special Reference to the Enlarged Thymus and Its Differential Diagnosis, Proc. Internat. Assemb. Inter-State Post-Grad. M. A., North America (1929) **5**:206, 1930.

29. Hasley, C. K., and DeTomasi, R. Q.: Cinex Camera Studies of the Thymus in Infants and Children, J. Michigan M. Soc. **29**:25, 1930.

gastrium, less marked above the sternum. The lungs were clear and the mediastinal dulness not increased. The roentgenogram of the chest showed forward displacement of the trachea at the level of the fifth and sixth cervical vertebrae, with a rather sharp bend. The possibility of a tumor in this region was considered likely. A study with barium sulfate of the esophagus showed it to be normal. Bronchoscopic examination confirmed the forward bending of the trachea but showed no evidence of tracheal compression. The larynx and esophagus were entirely normal. The absence of endoscopic evidence of tracheal or esophageal compression raised considerable doubt that the respiratory disturbance had originated in a tumor. The pediatrician in charge made the following note in discharging the patient: "The marked retraction in the epigastrium with relatively little retraction above the sternum suggests the possibility of (1) a soft sternum or (2) a congenital anomaly of the ligamentous attachments of the sternum—conditions which will lead to the development of a funnel chest." No irradiation was advised. A letter just received from the mother states that the child has "entirly overcome his breathing difficulty and is robust and healthy."

CONCLUSION

Chronic stridor in infancy, like chronic hoarseness, requires a careful direct study of the larynx. In the majority of cases the cause will be found within the larynx itself in the form of a flaccid superior aperture, an overdeveloped flabby epiglottis, palsy of one or both vocal cords, a tumor, web, cyst or hypertrophy of the subglottic tissues or ventricular bands. The tendency to ascribe the respiratory disturbance to extrinsic causes, particularly thymic pressure, solely on the basis of suspicious roentgenologic changes, without direct laryngoscopic examination, is still common enough in pediatric practice to impose on laryngologists the duty of urging consultation on all such problems. With proper diagnosis the possibilities of successful management are excellent enough to permit a favorable outcome in the majority of instances.

CHEMOPROPHYLAXIS AGAINST IMPENDING POLIOMYELITIS

REA E. ASHLEY, M.D.
SAN FRANCISCO

No acute infectious disease has ever been studied with greater intensity than acute anterior poliomyelitis, and yet, despite the great accumulation of experimental data, no positive method of prevention and no promising method of treatment has thus far been found. While this paper deals primarily with the application of chemicals to the olfactory nerves as a preventive measure against the disease, it is of interest to review some of the history and the experimental data which may lead to success in this method of prophylactic treatment.

Poliomyelitis has been a recognized entity since Joerg [1] described it in 1816. Heine, between the years 1840 and 1860, collected cases of a definite type from the records of paralysis of children and raised this type, the flaccid, atrophic spinal type, to a definite clinical entity. Medin, by 1890, had founded the symptomatology of the disease, and Wickman, by his clinical studies and his excellent histologic work, added important contributions to knowledge.

Experimental studies began in 1909, when Landsteiner and Popper [2] discovered that the disease could be transmitted to certain species of monkeys. In the same year Flexner and Lewis [3] in America and Landsteiner and Levaditi [4] in Europe demonstrated the infecting agent to be a filter-passing virus. That the nasal membrane affords a portal of entry for the production of experimental poliomyelitis in monkeys was demonstrated by Leiner and Von Wiesner [5] in 1910, and the ability of the virus to pass through the nonirritated nasal membrane was shown by Levaditi and Danulesco [6] in 1912.

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1. Joerg, J. C. G.: Ueber die Verkrümmungen des menschlichen Körpers und eine rationelle und sichere Heilart derselben, Leipzig, Mitzky u. Comp., 1810, p. 85.

2. Landsteiner, J., and Popper, E.: Ztschr. f. Immunitätsforsch. u. exper. Therap. **2**:377, 1909.

3. Flexner, S., and Lewis, P. A.: The Nature of the Virus of Epidemic Poliomyelitis, J. A. M. A. **53**:2095 (Dec. 18) 1909.

4. Landsteiner, K., and Levaditi, C.: Compt. rend. Soc. de biol. **67**:592, 1909.

5. Leiner, C., and von Wiesner, R.: Wien. med. Wchnschr. **60**:2482, 1910.

6. Levaditi, C., and Danulesco, V.: Compt. rend. Soc. de biol. **72**:342, 1912.

CHARACTERISTICS OF THE VIRUS

The size of the virus was estimated by Schultz and his co-workers [7] to be between 50 and 25 millimicrons, or from five to ten diameters smaller than the smallest body which can be seen by direct microscopic examination. The virus is resistant to glycerin, phenol,[8] acetone, ether, gastric juice, intestinal secretions and the like. It resists complete desiccation for at least thirty days. While roentgen rays seem to have no effect on the virus,[9] radiation from a cold quartz lamp inactivates it in less than three minutes. Oxidizing agents, such as solution of hydrogen peroxide and potassium permanganate, promptly inactivate it.[10] Olitsky [11] succeeded in growing the virus on nerve tissue of a human embryo, but thus far efforts to cultivate the organism on lifeless culture mediums have failed. The virus is highly selective, not only as to the host but as to particular nerve cells within the host.

PATHOGENICITY

The pathogenicity of the organism is restricted to man and certain species of monkeys, and even monkeys are difficult to infect by the intranasal route. Schultz [12] was unable to develop the disease in monkeys after placing them in direct contact with incubational or sick animals. Exposure for four days to an atmosphere more or less constantly charged with vaporized suspension of the virus did not lead to infection in his experiments. Schultz and Gebhardt,[13] however, were able to infect 95 per cent of the monkeys inoculated with virus by the intranasal route after a previous irrigation of the nasal membrane with an acid buffer solution. The incubation period of the natural disease is not known but is probably not less than six days nor more than twenty.

IMMUNITY IN POLIOMYELITIS

Immunity which follows the infection of poliomyelitis is positive and in most cases permanent. It is well known that some three quarters of the adult population has an active immunity to the disease if the

7. Schultz, E. W., and Krueger, A. P.: Proc. Soc. Exper. Biol. & Med. **26**: 600, 1929. Clifton, C. E.; Schultz, E. W., and Gebhardt, L. P.: J. Bact. **22**: 7, 1931; unpublished data.

8. Rhoads, C. P.: J. Exper. Med. **49**:701, 1929.

9. Lenz, M., and Jungeblut, C. W.: Proc. Soc. Exper. Biol. & Med. **29**: 814, 1932.

10. Schultz, E. W.: J. Pediat. **1**:25, 1932.

11. Olitsky, P. K., and Sabin, A. B.: Proc. Soc. Exper. Biol. & Med. **34**: 357, 1936.

12. Schultz, E. W.: Recent Advances in the Study of Poliomyelitis, J. Pediat. **1**:358 (Sept.) 1932.

13. Schultz, E. W., and Gebhardt, L. P.: Proc. Soc. Exper. Biol. & Med. **30**: 1010, 1933.

presence of neutralizing antibodies in the blood is an index. Thus far, however, no method has been found which will consistently confer immunity to infection by the intranasal route in monkeys. Serums have been of little or no value, and vaccines have not proved significantly effective against the infection.

PATHOLOGY OF THE DISEASE

The more evident pathologic characteristics of poliomyelitis have been known for years as a result of studies on human material and observation of the lesions found in experimentally infected monkeys. The characteristic lesion is destruction of the ganglion cells in the gray matter of the cord with consequent neurophagia of the destroyed nerve cells and more or less perivascular infiltration with round cells.

Fairbrother and Hurst,[14] Jungeblut and Spring [15] and Faber and Gebhardt [16] made important additional contributions to the pathology of the disease, all of which bear on the route by which the virus finds its way to the various levels of the central nervous system. Working with monkeys, these investigators made observations which seem to show conclusively that, contrary to the earlier views, which held that the virus was carried to and distributed within the central nervous system by the lymphatics, the blood stream or the cerebrospinal fluid, the virus in reality travels exclusively along nerve paths to the olfactory bulb and from there can travel to the cord by way of the motor fibers of the pyramidal tracts.

Faber [17] was able to correlate the experimental findings in monkeys with the clinical findings in human beings and point out that poliomyelitis is essentially a disease of the nervous system, throughout its entire course.

EPIDEMIOLOGY OF THE DISEASE

That the disease is spread chiefly, if not entirely, by the dissemination of droplets of the virus from the nasopharynges of carriers is well established.[18] Aycock [19] has pointed out the possibility of dissemination

14. Fairbrother, R. W., and Hurst, E. W.: J. Path. & Bact. **33**:17, 1930.

15. Jungeblut, C. W., and Spring, W. J.: Proc. Soc. Exper. Biol. & Med. **27**: 1076, 1930.

16. Faber, H. K., and Gebhardt, L. P.: J. Exper. Med. **57**:933, 1933.

17. Faber, H. K.: Medicine **12**:83, 1933.

18. Flexner, S., and Lewis, P. A.: Epidemic Poliomyelitis in Monkeys: A Mode of Spontaneous Infection, J. A. M. A. **54**:535 (Feb. 12) 1910. Lucas, W. P., and Osgood, R. B.: Transmission Experiments with the Virus of Poliomyelitis: Finding the Virus in the Nasal Secretion of a Human Carrier Four Months After the Acute Stage of a Second Attack of Poliomyelitis, ibid. **60**: 1611 (May 24) 1913. Flexner, S.; Clark, P. F., and Fraser, F. R.: Epidemic

(Footnotes continued on next page)

of the virus by such vehicles as milk, but many investigators [20] have found it exceedingly difficult to infect monkeys by feeding them the virus. Schultz fed 2 monkeys enough ground cord infected with the virus, suspended in milk, to infect more than 500,000 monkeys by the intracerebral route. Not only did the disease fail to develop, but the monkeys failed to become immune. Clark, Roberts and Preston [21] were unable to produce poliomyelitis either by oral administration or by injection of as much as 10 cc. of the suspension of the virus into a loop of the small intestine.

Erber and Pettit [22] and Levaditi and his co-workers [23] found that poliomyelitis developed in a rather high percentage of monkeys after intracutaneous injection of a suspension of spinal cord infected with the virus. Trask and Paul [24] recently repeated and confirmed this work with freshly isolated human strains of virus. These findings were not remarkable, however, since it is generally understood that any method whereby the virus is forcefully introduced into the tissue may result in infection.

The entire rationale of chemoprophylaxis depends on the correctness of the hypothesis that the usual portal of entry of the virus is the olfactory area. If it is proved that this is not usually the case, chemoprophylaxis is not likely to have practical value.

Time does not permit a review of all the evidence on this point, but in favor of the intranasal route are the following points brought out by Faber: [25]

1. There is only one area in man where any part of the central nervous system is directly exposed on the surface of the body, and this is the so-called olfactory epithelium. The hairs of the olfactory cells are by no means entirely embedded in the nasal mucosa of the surface, but a large proportion of them actually lies free on the surface directly exposed to the air, an arrangement essential to the sense of smell.

Poliomyelitis: Passive Human Carriage of the Virus of Poliomyelitis, ibid. **60**: 201 (Jan. 18) 1913.

19. Aycock, W. L.: Am. J. Hyg. **8**:791, 1927.

20. Flexner, S., and Lewis, P. A.: J. Exper. Med. **12**:227, 1910. Amoss, H. L., in Rivers, T. M.: Filterable Viruses, Baltimore, Williams & Wilkins Company, 1928, p. 174. Schultz, E. W.: Proc. Soc. Exper. Biol. & Med. **26**:632, 1929. Clark, P. F.; Schindler, J. T., and Roberts, D. J.: J. Bact. **20**:213, 1930.

21. Clark, P. F.; Roberts, D. J., and Preston, W. S.: J. Prev. Med. **6**: 47, 1932.

22. Erber, B., and Pettit, A.: Compt. rend. Soc. de biol. **117**:1175, 1934.

23. Levaditi, C.; Kling, C., and Haber, P.: Bull. Acad. de méd., Paris **115**: 431, 1936.

24. Trask, J. D., and Paul, J. R.: J. Bact. **31**:527, 1936.

25. Faber, H. K.: Am. J. Pub. Health **23**:1024, 1933.

2. The nasal mucosa is the only portion of the surface of the body where infection with poliomyelitis may be produced with regularity and without trauma.

3. Other surfaces, such as that of the gastrointestinal tract, have been shown to be invulnerable to the virus.

4. Infection is accomplished with great difficulty by way of the blood stream.

5. Active virus has been detected in the nasal mucosa in patients with the disease, in carriers and in one instance in a person who five days later came down with the disease.

6. The virus has particular selectvitiy for nerve tissue, and bringing virus into direct contact with nerve tissue is the only method reasonably certain to produce the disease.

7. The olfactory cells in the nasal mucosa are in reality nerve cells connected by their axons directly with the central nervous system.

Other evidence strongly supporting this view is the fact that the terminal processes, the olfactory hairs and the olfactory nerves themselves are unmyelinated, which renders them susceptible to infection.

Finally, Schultz and Gebhardt [26] severed the olfactory tracts in 9 monkeys and tried to infect them by the intranasal route; the disease did not develop in any of the 9, while in an equal number of controls it developed within seven days. Sixteen days later the same monkeys were tested in a similar manner, and the disease did not develop, while in 2 control monkeys it developed in eight days. The 9 monkeys were then inoculated intracranially and typical poliomyelitis developed in all of them in from five to eight days, which showed their susceptibility to the virus. Brodie and Elvidge [27] performed similar experiments with similar results.

The reasons given and the experimental results described strongly support the view that the normal portal of entry is the olfactory nerve.

CHEMOPROPHYLAXIS

The idea that certain chemicals applied to the nasal mucous membrane might make it less permeable to the virus of poliomyelitis occurred to three different groups of investigators at about the same time. It had been known for several years that certain chemicals sprayed on the nasal mucosa of mice made them increasingly resistant to other virus disease, particularly vaccinia, encephalitis (the St. Louis type) and

26. Schultz, E. W., and Gebhardt, L. P.: Proc. Soc. Exper. Biol. & Med. **31**: 728, 1934.

27. Brodie, M., and Elvidge, A.: Science **79**:235, 1934.

equine encephalomyelitis.[28] On the basis of the assumption that the virus does enter through the olfactory area of the nasal tract, attempts were made by these several investigators to effect blocking of this portal of entry by local application of chemicals to the nasal mucosa of monkeys.

In 1935 Armstrong and Harrison[29] reported that the application of a 4 per cent solution of alum in the nostrils of monkeys tended to prevent the disease when active virus was subsequently instilled into the nose. Shortly thereafter Schultz and Gebhardt[30] confirmed this characteristic of chemicals and eventually found that a 1 per cent solution of zinc sulfate (chemically pure) gave a more lasting and a higher degree of protection than any other chemical of a long list. A single spraying of 1 per cent zinc sulfate was sufficient to protect monkeys against the virus for at least one month after repeated intranasal instillation.

The protection is thought to be due to a change in the permeability of the mucous membrane or of the nerve endings rather than to any direct action of the chemical on the virus. Schultz[31] has suggested that the possible mode of action may be some transient union of the zinc ion with the protoplasm of the nerve cells, which alters their permeability to the virus. He stated that destruction of the olfactory endings, even of the olfactory cells themselves, cannot be entirely excluded as yet. He is at present engaged in studying this question.

This experimental work of Armstrong and Schultz offered the first definite hope of controlling infantile paralysis, since immune serums and vaccines which had been advocated as a protection in human beings had completely failed to protect monkeys against the inoculation of the virus. Their work also paved the way for trials of chemicals in preventing the disease in human beings.

As Schultz has stated, there are three important questions which must be answered:

1. Is this type of prophylaxis which is so effective for monkeys applicable to man?

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28. Ledingham, J. C. G.: Brit. J. Exper. Path. **8**:12, 1927.

29. Carnot, P.; Camus, L., and Benard, H.: Compt. rend. Soc. de biol. **95**:457, 1926. Le Fèvre de Arric, M.: ibid. **96**:208, 1927. Rivers, T. M.; Stevens, H., and Gates, F. L.: J. Exper. Med. **47**:37, 1928. Armstrong, C.: Pub. Health Rep. **48**:1, 1933. Olitsky, P. K., and Cox, H. R.: Science **80**:566, 1934. Armstrong, C., and Harrison, W. T.: Pub. Health Rep. **50**:22, 1935.

30. Schultz, E. W., and Gebhardt, L. P.: Proc. Soc. Exper. Biol. & Med. **34**: 133, 1936. Schultz, E. W.: ibid. **35**:524, 1937. Schultz, E. W., and Gebhardt, L. P.: Zinc Sulfate Prophylaxis in Poliomyelitis, J. A. M. A. **108**:2182 (June 26) 1937.

31. Schultz, E. W.: Future of Chemoprophylaxis as a Means for Practical Control of Poliomyelitis, unpublished data.

2. Can the mechanical difficulties of applying such prophylactic measures be overcome sufficiently to make it a practical public health procedure?

3. Are there any serious contraindications for general use of such prophylactic measures?

EFFECT OF CHEMICALS ON THE NASAL MUCOSA OF HUMAN BEINGS

In an effort to answer these questions, Peet, Echols and Richter [32] performed experiments to determine the effect of zinc sulfate on the nasal mucous membrane of human beings. For these studies a group of student volunteers were tested. Peet found that zinc sulfate when sprayed on the nasal mucosa produced a severe burning sensation, with coryza and in most cases a severe headache which lasted for several hours. These discomforting symptoms were almost entirely eliminated when a 1 per cent solution of pontocaine hydrochloride was added to the zinc sulfate solution. Some of the students, however, complained of a dull headache of from one to several hours' duration after the initial effect of the anesthetic had disappeared. In these studies, children were much less adversely affected than were adults. Peet and his co-workers next took up the problem of the effect of zinc sulfate on the sense of smell. Their studies showed that the sense of smell is temporarily lost but returns to normal in from one to two weeks in all cases.

Echols and his co-workers [33] tested 11 medical students and verified Peet's work. The average duration of the loss of sense of smell in their experiments was eleven days. (Other investigators, however, have found that the anosmia persists for a much longer time if the solution is instilled rather than sprayed into the nostrils.[34]) Inspection of the nasal membrane following spraying disclosed no edema, hyperemia or other evidence of trauma or inflammation.

Schultz [35] has studied the reaction of 1 per cent zinc sulfate on the mucous membrane of monkeys microscopically and has noted varying degrees of acute inflammation in the olfactory membrane and some desquamation of the olfactory epithelium in specimens removed twenty-four to forty-eight hours after treatment. He has, however, occasionally seen both these conditions in specimens of untreated mem-

32. Peet, M. M.; Echols, D. H., and Richter, H. J.: Chemical Prophylaxis for Poliomyelitis: Technic of Applying Zinc Sulfate Intranasally, J. A. M. A. **108**:2184 (June 26) 1937.

33. Echols, D. H.; Richter, H. J., and Peet, M. M.: Univ. Hosp. Bull., Ann Arbor **3**:32, 1937.

34. Shahinian, L.: Unpublished data.

35. Schultz, E. W.: Unpublished data.

branes. Unmistakable evidence of regeneration of olfactory epithelium in several monkeys killed forty-eight hours after treatment with zinc sulfate has recently been demonstrated by Schultz, and, while the experiments have not yet been completed, his observations thus far indicate that so far as the ordinary epithelial covering is concerned regeneration is rapid. He is at present concerned with what happens to the olfactory cells.

Schultz has emphasized that, whatever the mechanism in protection may be, restoration is effected, since the majority of animals eventually become susceptible again. In an unpublished paper, "Future of Chemoprophylaxis as a Measure for Practical Control of Poliomyelitis," recently read before the Academy of Medicine at Portland, Ore., he suggested the possibility of actual regeneration of olfactory nerve endings and stated: "A regeneration of the peripheral processes would not be novel, but evidence that destroyed neurons are replaced in some way would indeed be new."

While these experiments are not entirely conclusive, they do indicate that 1 per cent zinc sulfate causes no permanent damage to the olfactory mucosa or nerves.

METHODS OF APPLICATION OF CHEMICALS

In the attempt to answer the question, "Can mechanical difficulties be overcome sufficiently to make treatment a practical public health measure?" two general methods of applying chemicals to the olfactory area have been described, the atomizer method and the postural method.

Peet, Echols and Richter made a complete study of the first group and determined the ordinary atomizer to be inefficacious. They described placement of the chemical by the use of a special atomizer tip introduced between the middle turbinate and the septum. This procedure is difficult, especially with small children, and is not free from danger. Meyerson [36] showed that among children a maximum of 25 per cent could be effectively treated by this method.

Postural methods depend on the localization of the prophylactic fluid by gravitation. Pentecost [37] advises introducing the solution into the nasal cavity with an olive-tipped urethral catheter placed between the middle turbinate and the septum with the patient in the Proetz position so that the head is inverted with the chin and the external auditory meatus in the same vertical plane. He treated approximately 100 children 8 years of age or older by this method and obtained immediate 100 per cent anosmia. No serious after-effects occurred.

36. Meyerson: Personal communication to the author.

37. Pentecost, R. S.: Canad. Pub. Health J. **28**:493, 1937.

SHAHINIAN'S METHOD

Shahinian, Bacher, McNaught and Newell [38] at Stanford recently described a simple and efficient method of applying solutions to this area. They have shown that if the head is completely extended and inverted so that Reid's base line is parallel to the floor the cribriform plate will be in approximately the same horizontal plane and liquids dropped slowly into the nose will automatically cover the olfactory area completely. Their experiments were done with radiopaque solutions. Roentgenograms showed that in every case the solution automatically gravitated to the olfactory area regardless of the age of the patient or of any malformations of the septum or turbinates. Shahinian also measured the amount of liquid necessary to immerse the olfactory area completely. Although there is some variance between different persons, his experiments show that, strangely, children under 10 years require 0.5 cc. of fluid and children between 10 and 14 years require but 0.4 cc., while adults require only 0.25 cc. He stressed the quantity of fluid required because he felt that many of the distressing symptoms are due to the solution's overflowing into the sinuses. To remove the fluid, the patient is instructed to turn over to the prone position, lift the head and gently blow the nose.

Meyerson recently described a similar placement method before the Eastern Section of the American Laryngological, Rhinological and Otological Society.

Here again the experimental evidence is not absolutely conclusive, but it would seem that the postural method described by Shahinian and Meyerson overcomes most of the mechanical difficulties of application. By this method large numbers of persons can be treated quickly and effectively by any physician or public health worker after a little instruction.

PRACTICAL APPLICATION OF CHEMOPROPHYLAXIS FOR HUMAN BEINGS

The question, "Is this method of treatment applicable to man?" can be answered only by a completely satisfactory field test. Thus far chemoprophylaxis in human beings has been tested during two epidemics; in neither case have the results been informative.

In the Alabama epidemic of 1936, great numbers of persons had their noses sprayed with a mixture of trinitrophenol and alum by means of ordinary atomizers, but, unfortunately, no control of the method, no record of the number of times each person was given the spray and

38. Shahinian, L.; Bacher, J. A.; McNaught, R. C., and Newell, R. R.: Chemoprophylaxis in Poliomyelitis: The Technic of Applying Chemical Agents to the Olfactory Mucosa, J. A. M. A. **110**:1254 (April 16) 1938.

no accurate estimate of the efficiency of the spraying was possible. Armstrong [39] after a critical study of this field test came to the conclusion that the value of the solution could not be determined because of the faulty method of application.

A second opportunity of testing the value of chemical sprays as a preventive measure came during the Toronto epidemic of 1937. Tissdell and his associates [40] reported the method of treatment and the results. In this epidemic the solution used was 1 per cent zinc sulfate U. S. P., 0.5 per cent pontocaine hydrochloride in 0.5 per cent saline solution. The spraying was done entirely by otolaryngologists and was given on two occasions with an interval of from ten to twelve days between sprayings. Fifty-two hundred children between 3 and 10 years had their noses sprayed once, and of these 4,600 received a second spraying. Sixty-three hundred children of approximately the same ages were used as controls. Some 20 per cent of the treated children were tested for anosmia, and only about 14 per cent of these showed impairment or loss of the sense of smell. No tests were made of the degree or the duration of the impairment. No appreciable difference was found in the incidence of poliomyelitis in the group treated by spraying and in the control group. The report concludes that since the spraying must be done by otolaryngologists or other persons especially trained in intranasal treatment and cannot be instituted quickly enough to prevent the disease during an epidemic, it cannot be considered a practical public health measure.

While there is no information available regarding the relation of anosmia which follows chemical treatment of the olfactory tract to the degree of protection produced, it seems reasonable to suppose that the more complete the anosmia the more olfactory nerve endings are blocked and the less apt is the virus to reach the olfactory nerves. Assuming this relation to exist, the ineffectiveness of treatment with zinc sulfate in the Toronto epidemic is easily explainable; since anosmia developed in only 14 per cent of the children whose noses were sprayed, it follows that in 86 per cent the solution was not adequately applied to the olfactory areas. Schultz now emphasizes the importance of using functional tests for anosmia as an index to determine when second treatments are due during an epidemic rather than basing the decision on the duration of the protection afforded in animals. While the experience during human epidemics has so far failed to show any benefit derived from the spraying of chemicals, further tests should be made with the postural method of placement before the procedure is condemned.

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39. Armstrong, C.: Am. J. Pub. Health **27**:103, 1937.

40. Tissdell, F. F.; Brown, A., and Defries, R. D.: Canad. Pub. Health J. **28**:523, 1937.

SUMMARY AND COMMENT

1. The organism of poliomyelitis is a small, filter-passing virus, which is highly selective for nerve tissue and which progresses within the axis-cylinders of the nerves.

2. The olfactory nerve is the most logical and probably the only normal portal of entry.

3. Serums and vaccines have failed to protect monkeys against the disease.

4. Certain chemicals applied to the olfactory tracts of monkeys have rendered them resistant to infection with the virus after repeated intranasal instillation. Fifty or sixty chemicals have thus far been tested for this protective property, and while zinc sulfate is not toxic and seems to be the most effective found, it causes certain irritating and unpleasant symptoms and is not the ideal solution.

5. Anosmia which develops after chemical treatment is probably an evidence of the completeness of the nerve blocking and an indication of the protection. The duration of the anosmia when zinc sulfate is used varies greatly in different persons, but apparently the sense of smell always returns.

6. If this type of treatment is practical, the effectiveness of the protection will depend on the thoroughness of the nerve-blocking. Functional tests for the return of the sense of smell should be the guide for further chemical application.

7. The treatment can best be applied by the postural method described by Shahinian and Meyerson; it is simple and efficient, and by means of it treatment can be given quickly to large groups.

8. While the experience during human epidemics has so far failed to show definite protection, further field tests are indicated, since previous methods of application have been faulty.

9. Should this treatment prove effective in man, it will still be far from an ideal prophylactic, chiefly because of the short duration of the protection afforded, and the failure of the method to produce any immunity in the individual to the virus. Nevertheless, it is the only method which at present promises any hope of actually combating the disease.

IRRITATION OF THE THROAT FROM CIGARET SMOKE

A STUDY OF HYGROSCOPIC AGENTS

HOWARD C. BALLENGER, M.D.
CHICAGO

The laboratory experiments of Mulinos and Osborne [1] on the conjunctivas of rabbits and the clinical investigation of Flinn [2] on the respiratory mucosa led to their conclusion that diethylene glycol is definitely less irritating than is glycerin when used as the hygroscopic agent in cigarets. Haag,[3] at the Medical College of Virginia, in duplicating the work of Mulinos and Osborne failed to find any difference in irritation of the conjunctival sacs of rabbits as determined by the appearance of hyperemia, edema and blepharospasm and by the objection of the animal. Holck and Carlson,[4] at the University of Chicago, failed to find difference in salivation in human beings subjected to the smoke of the two types of cigarets. From a clinical standpoint, Sharlit [5] was unable to find any difference between the irritating properties of cigarets treated with glycerin and of those treated with diethylene glycol. In a clinical investigation in 1936.[6] Johnson and I came to the same conclusion.

The clinical investigation reported here was planned along the general lines of the one which formed the basis of Flinn's [2] paper. The

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From the Department of Otolaryngology, School of Medicine, Northwestern University.

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1. Mulinos, M. G., and Osborne, R. L.: Pharmacology of Inflammation: Influence of Hygroscopic Agents on Irritation from Cigarette Smoke, Proc. Soc. Exper. Biol. & Med. **32**:241 (Oct.) 1934; Irritating Properties of Cigarette Smoke as Influenced by Hygroscopic Agents, New York State J. Med. **35**:590 (June 1) 1935.

2. Flinn, F. B.: Some Clinical Observations on the Influence of Certain Hygroscopic Agents in Cigarettes, Laryngoscope **45**:149 (Feb.) 1935.

3. Haag, H. B.: Studies on the Physiologic Action of Di-Ethylene Glycol: The Effect upon the Irritating and Toxic Properties of Cigaret Smoke, J. Lab. & Clin. Med. **22**:341 (Jan.) 1937.

4. Holck, H. G. O., and Carlson, A. J.: Alleged Influence of Glycerine and Diethylene Glycol upon the Irritating Qualities of Cigarette Smoke, Proc. Soc. Exper. Biol. & Med. **36**:302 (April) 1937.

5. Sharlit, H.: Cigarette Smoke as a Health Hazard, New York State J. Med. **35**:1159 (Nov. 15) 1935.

6. Ballenger, H. C., and Johnson, V. H.: Effects on Throat and Conjunctiva of Hygroscopic Agent Used in Cigarets, Arch. Otolaryng. **25**:75 (Jan.) 1937.

results are not in agreement with Flinn's report and taken in conjunction with the reported work of Haag and of Holck and Carlson, as well as the clinical investigation by Johnson and me, indicate that the current status of scientific knowledge of the relative irritating effects of diethylene glycol and glycerin when used in cigarets does not justify the conclusion that these two hygroscopic agents present any important difference in this specific property.

The present investigation is a continuation of that done in 1936. As in the first investigation, three lots of cigarets were prepared (lots 1, 2 and 3), of which one (lot 1) contained diethylene glycol (2.68 per cent) as the hygroscopic agent, another (lot 3) was moistened with glycerin (2.69 per cent) and the third (lot 2), the control cigarets, had no hygroscopic agent of any kind. Otherwise the cigarets were identical, all having been made by one tobacco manufacturer of the same batch of tobacco. The presence of diethylene glycol in the cigarets of lot 1 and of glycerin in the cigarets of lot 3 was established by the isolation and qualitative identification of these compounds.

Ten well known otolaryngologists were invited to select a group of cigaret smokers; the majority of those selected (74 per cent) seemed to have symptoms from smoking.

The identity of the various types of cigarets was not known to any of the examiners connected with the tests or to the subjects until after the investigation had been completed and the final reports had been made.

The 102 subjects (table 1) on whom the present tests were conducted varied in age from 17 to 62 (average about 30). They had been smoking an average of about twenty cigarets a day for an average of twelve years and continued to smoke the same number during the period of the tests.

METHOD OF STUDY

The tests were conducted in two stages. The first lasted for three weeks, in which cigarets from the various lots were issued for one week to each subject. At the end of each week he returned to the examiner for examination and report of symptoms, if any, at which time a new lot was issued. The cigarets were rotated in such a manner that at any given time one third of the subjects were smoking cigarets from lot 1, another third from lot 2 and the final third from lot 3. This was done to equalize any effects of climatic changes or epidemics of colds. Any objective signs or subjective symptoms of irritation were noted.

The second phase of the test was purely subjective; that is, each smoker was given cigarets from all three lots for a week. He smoked them in any manner he chose in an effort to determine any difference in irritation or symptoms from the three types. The conclusions from both tests were noted and tabulated.

SYMPTOMS FROM SMOKING

The various symptoms attributed to smoking which the subjects presented prior to the tests (table 1), listed in the order of their frequency of occurrence, were: a dry or irritated throat (36); coughing, usually dry and worse in the morning (34); husky or hoarse voice (15); excess secretion, usually mucus (13). Symptoms referable to the nose, such as stuffiness, dryness, or sneezing, were mentioned in a few (10) instances. Various complaints mentioned by only 1 or 2 subjects each were tickling of the throat, burning of the tongue, "dopey" feeling, burning of the eyes, fatigue of the eyes, bad taste, nervousness and loss of appetite.

The amount of smoking and the presence of symptoms seem to have some relation, especially in the case of the morning cough which raises more or less dark mucus (table 2). The total number of symptoms attributed to smoking is higher for the heavy smokers than for the light smokers. However, secretion of mucus in the nose or pharynx, stuffy or irritated feeling in the nose, dry or irritated sensation in the mouth or pharynx and husky or hoarse voice do not seem to have much relation to the number of cigarets smoked per day.

OBJECTIVE EXAMINATION

An effort was made in each case by each examiner to determine by careful objective examination the amount of irritation produced during the test periods. This examination consisted of inspection of the mucosa of the nose, throat and larynx. Notations were made of changes in the secretions, dryness, granulations, thickness and color. The color was charted after comparison with and in accordance to the Tallqvist scale.

Five examiners stated that no difference between the effects of the two lots could be ascertained. Three examiners found that in the few instances in which changes were noted they were so evenly divided as to be without significance. The two examiners who reported differences based on objective examination were not in agreement as to which cigaret produced the more irritation. One found more irritation from the diethylene glycol cigarets and the other from the glycerin cigarets. Of the 102 subjects included in these tests, 75 were found to have no unfavorable changes in the mucosa of the nose, throat or larynx as indicated by the regular periodic examinations. In 9 subjects the most unfavorable results followed the use of the untreated cigarets. In 8 the most adverse reactions followed the use of the cigaret treated with diethylene glycol. In 7 the most unfavorable results followed the use of the cigarets treated with glycerin. In 2 instances, in which the control cigarets were found to cause the least irritation, no difference was observed between the effect of the other two. In 1 subject, for whom the diethylene glycol cigarets were found the least irritating, no

TABLE 1.—*Symptoms Attributed to Cigaret Smoking by 102 Subjects*

Subject No.	Age	No. Smoked per Day	No. of Years of Smoking	Brand	Hygroscope Agent	Symptoms Attributed to Smoking
		(A) Subjects who smoked more than 20 cigarets daily				
1	45	40	20	A	Glycerin	Cough
2	31	40	20	B	Glycerin	Dry throat and nose, cough
3	40	35	20	B	Glycerin	Cough (chronic)
4	30	35	10	C	Glycerin	
5	38	30	20	C	Glycerin	Dryness of throat, cough
6	62	30	20	C	Glycerin	Dry throat, cough
7	29	30	12	D	Glycerin	
8	25	30	4	C	Glycerin	Cough
9	45	30	30	B	Glycerin	Tickly throat, huskiness, cough
10	27	27	20	E	Diethylene glycol	Stuffy nose, dry throat, cough
11	31	25	15	C	Glycerin	Dry throat and dry cough
12	53	25	38	F	Glycerin	Dry throat, husky voice, cough
13	28	25	10	B	Glycerin	
14	31	25	15	E	Diethylene glycol	Dry cough, postnasal drip
15	31	25	15	B	Glycerin	Mucus in throat, cough (wet?)
16	26	25	8	E	Diethylene glycol	
17	26	25	8	E	Diethylene glycol	
18	28	25	10	G	Glycerin	Dry throat
19	17	23	2	B	Glycerin	
		(B) Subjects who smoked 20 cigarets daily				
20	40	20	10	G	Glycerin	Soreness of throat
21	28	20	7	H	Glycerin	Dryness of nose and throat
22	25	20	5	B	Glycerin	Dryness, bad taste
23	43	20	20	I	Glycerin	Dry nose, cough
24	29	20	10	C	Glycerin	Stuffy nose, dry throat, cough
25	35	20	15	F	Glycerin	
26	30	20	15	C	Glycerin	Dry throat, cough, postnasal drip
27	..	20	10	C	Glycerin	
28	40	20	..	H	Glycerin	Dry throat, cough
29	37	20	..	H	Glycerin	Dry throat, mucus in throat
30	46	20	..	B	Glycerin	Cough
31	28	20	..	B	Glycerin	
32	..	20	..	E	Diethylene glycol	Irritated throat, postnasal drip
33	26	20	9	H	Glycerin	Husky voice
34	33	20	13	B	Glycerin	Dry throat, cough, irritation when speaking
35	29	20	12	H	Glycerin	Cough (occasional)
36	28	20	12	D	Glycerin	Raw throat
37	37	20	16	H	Glycerin	Dry throat
38	30	20	10	H	Glycerin	Nervousness
39	32	20	15	E	Diethylene glycol	Irritated throat, "dopey" feeling
40	37	20	14	B	Glycerin	"Dopeyness"
41	39	20	18	H	Glycerin	Secretion in throat
42	41	20	18	F	Glycerin	
43	30	20	10	B	Glycerin	Cough
44	56	20	40	J	Glycerin	Cough
45	23	20	4	B	Glycerin	
46	29	20	5	E	Diethylene glycol	Cough
47	25	20	20	E	Diethylene glycol	Dry throat
48	30	20	18	B	Glycerin	Irritated throat
		(C) Subjects who smoked fewer than 20 cigarets daily				
49	32	18	12	I	Glycerin	
50	20	18	5	E	Diethylene glycol	
51	32	18	14	C	Glycerin	Cough (occasional)
52	30	15	10	E	Diethylene glycol	
53	44	15	15	B and H	Glycerin	Raw, burning throat, cough
54	32	15	10	H	Glycerin	
55	39	15	18	A	Glycerin	Bad taste
56	34	15	14	C	Glycerin	Cough
57	30	15	10	C	Glycerin	Irritated throat
58	27	15	10	C	Glycerin	Sneezing, cough
59	35	15	13	C	Glycerin	Irritated throat, eye fatigue
60	31	15	15	K	Glycerin	Cough
61	31	15	12	B	Glycerin	Dry throat, cough
62	36	15	15	E	Diethylene glycol	Stuffy nose, dry throat, cough

TABLE 1.—*Symptoms Attributed to Cigaret Smoking by 102 Subjects—Continued*

Subject No.	Age	No. Smoked per Day	No. of Years of Smoking	Brand	Hygroscope Agent	Symptoms Attributed to Smoking
63	28	15	12	H	Glycerin	Cough
64	24	15	8	L	Glycerin	Cough
65	32	15	10	C	Glycerin	
66	..	15	6	B	Glycerin	Loss of voice (occasional)
67	24	15	7	C	Glycerin	
68	29	15	9	M	Glycerin	
69	30	15	15	I	Glycerin	Dry throat and cough, perspiring hands
70	30	15	20	N	Glycerin	Hoarseness
71	35	15	15	C	Glycerin	Hoarseness, loss of appetite
72	38	15	20	D	Glycerin	Cough
73	27	15	5	B	Glycerin	
74	24	15	7	E	Diethylene glycol	
75	40	12	15	E	Diethylene glycol	Hoarseness
76	34	12	6	B	Glycerin	Burning tongue
77	29	12	7	E	Diethylene glycol	
78	48	10	10	H	Glycerin	
79	23	10	4	B	Glycerin	
80	26	10	3	A	Glycerin	Cough
81	24	10	4	C	Glycerin	Dry throat, cough
82	25	10	11	C	Glycerin	Hoarseness, cough, mucus in throat
83	30	10	12	B	Glycerin	Dry throat
84	25	10	8	B	Glycerin	Postnasal drip
85	28	10	6	C	Glycerin	Rough voice, postnasal drip
86	30	10	15	B	Glycerin	Dry throat, migraine
87	29	10	9	H	Glycerin	Irritated throat, husky voice
88	48	10	30	B	Glycerin	Cough
89	25	10	10	H	Glycerin	Irritated throat, cough, huskiness
90	22	10	7	H	Glycerin	
91	22	10	3	E	Diethylene glycol	Cough (occasional)
92	38	9	15	D	Glycerin	
93	29	8	10	O	Glycerin	
94	25	8	3	B	Glycerin	Occasional cough
95	24	7	4	A	Glycerin	Irritated throat
96	28	6	10	E	Diethylene glycol	
97	27	6	6	B	Glycerin	
98	24	5	6	L	Glycerin	
(D) Subjects for whom the number of cigarets smoked per day was not specified						
99	..	..	..	B	Glycerin	Irritated throat
100	30	.	..	E	Diethylene glycol	Irritated throat
101	48	..	..	B	Glycerin	Cough
102	..	.	..	..	.....	

TABLE 2.—*Symptoms Attributed to Smoking (198 Subjects)*

No. of Cigarets per Day	Number of Subjects	Percentage of Subjects					
		No Symptoms	Cough (Usually in the Morning)	Husky Voice	Excess Mucus	Dry or Irritated Throat	Dry or Congested Nose
5 to 14	38	28	21	7	6	20	0
15 to 24	119	24	25	13	8	27	5
25 to 40	41	18	32	9	7	21	5

difference was seen between the effect of the glycerin and the control cigarets. It would seem that these results indicate clearly that no objective evidence of irritation occurred in this investigation which could be attributed to the effect of either of the hygroscopic agents under investigation.

TABLE 3.—*Reactions to Cigarets Treated with Diethylene Glycol or with Glycerin* *

Subject No.	Order of Preference†		Subjective Reactions to Smoking (3 Week Period)			Objective Signs
	3 Week Period	1 Week Period	Diethylene Glycol-1	Glycerin 3	Controls 2	
	(A) Subjects who smoked more than 20 cigarets daily					
1	3-2 1	.....	Irritating to throat, cough	.....	Less irritating	None
2	3 1 2	3 1 2	Dry throat	.....	Dry throat	None
3	3 2 1	3 2 1	Irritated, dry nose	.....	Irritating	None
4	3-2 1	3 2 1	No taste	Same as C	Milder than C	None
5	3 1 2	1 3 2	.....	Mildest	Cough	None
6	2 1-3	2 1-3	Dryness	Dry tongue	.....	None
7	2-1 3	2-1 3	.....	Slightly irritating to throat	.....	None
8	3 1 2	1-3 2	Tastes better than 2	Least irritating	Irritating	None
9	1 2-3	1 2 3	Less irritating	More irritating	Cough (cold)	3 least 1 most
10	.....	1 2 3	.....	Bad taste	Dry throat (1 day)	None
11	.....	2 3 1	.....	.....	.....	None
12	1 3 2	1 3 2	.....	.....	Cough, tickling	None
13	3 2 1	3 2 1	Throat dry	Mild and best	Less irritating than 1	2 most
14	3 2 1	3 2-1	Irritation	Less irritation	Milder than 1	None
15	.....	.....	.....	.....	.....	None
16	1-3 2	1-3 2	.....	Better than 2	Dry throat	1 most
17	1 3 2	1 3 2	.....	.....	Irritating to throat	None
18	2 3 1	2 3 1	Harsh, strong	Slightly irritating	.....	2 most
19	1-2 3	1-2 3	.....	Irritating to throat, cough	.....	3 most
	(B) Subjects who smoked 20 cigarets daily					
20	2-3 1	.....	Discomfort in throat	Discomfort in throat	Cough, hoarseness	None
21	1 2-3	.....	Better than 3	Dry throat	Burning tongue	None
22	3 2 1	3 2 1	Dry throat; strong	Mild, better taste	Milder than B	None
23	1-3 2	1 3 2	.....	.....	Slight sting	None
24	3 2-1	1 3 2	Cold, secretion, redness	Less irritating	Less secretion	None
25	.....	1 2 3	Nasal secretion	.....	.....	None
26	3 1-2	3-1 2	Cough	Less irritating than 2	Throat better	None
27	2-3 1	2-3 1	Cough, hoarseness	Clearing throat	Better, same as C	None
28	1-2 3	2 1 3	.....	More irritating than 2	.....	None
29	1-3 2	1-3 2	.....	.....	Taste terrible	None
30	1-2 3	2 1 3	.....	More irritating than 2 (cold)	Less irritating than B	None
31	.....	.....	.....	.....	.....	None
32	1-3 2	1-3 2	.....	.....	Stinging	None
33	1-2 3	2 1 3	.....	Heartburn	.....	None
34	.....	1 3 2	.....	.....	Dry throat	2 most
35	2-3 1	2-3 1	Raw throat, cough	.....	.....	1 most
36	.....	1-2 3	Irritating to throat	Slight hoarseness	Irritating to throat	3 most
37	.....	1-3 2	.....	.....	.....	2 most
38	1 3 2	1 3 2	Least irritating; headache	Less irritating to mucous membrane	Dry nose, headache	None
39	1-3 2	1 3 2	.....	.....	More irritating	None
40	.....	1 2-3	.....	.....	.....	3 most
41	1 2 3	1 2 3	Less irritating	.....	Less irritating than 3	3 most
42	1-2 3	1-2 3	.....	More irritating	Most irritating	2 most
43	2 1 3	2 1 3	Irritating	Most irritating	Less irritating	1 most
44	1-2 3	2 1 3	Unsatisfactory	Tongue sore	Better than 1	None
45	1-3 2	1 3 2	.....	.....	Dry throat	None
46	3 2 1	3 2 1	Strong and irritating	Mild, less irritating	Better than 1 (cough)	None
47	2 3 1	2 3 1	Harsh, impossible	Slightly irritating	Better than 3	1 most
48	3 1-2	3 2 1	Bites	.....	Bites	2 most

* All subjects continued to smoke approximately the same number of cigarets they had used before the test.

† A dash between the numbers indicates no preference.

TABLE 3.—*Reactions to Cigarets Treated with Diethylene Glycol or with Glycerin *—Continued*

Subject No.	Order of Preference: 3 Week Period	Order of Preference: 1 Week Period	Subjective Reactions to Smoking (3 Week Period): Diethylene Glycol 1	Glycerin 3	Controls 2	Objective Signs
			(C) Subjects who smoked fewer than 20 cigarets daily			
49	.....	.....	.....	Did not like	Better than 1	None
50	.....	1 2 3	Mild	Stronger	Better than 1	None
51	1-2 3	2 1 3	.....	Dry throat	.....	None
52	3 1 2	3 1 2	.....	.....	.....	None
53	.....	.....	.....	.....	.....	None
54	.....	.....	.....	.....	.....	None
55	.....	2 1 3	Dry throat, cough	Irritating (cold)	Dry throat	None
56	1 2 3	1 2 3	Milder than 2	Irritating to throat (cold)	Less irritating	None
57	1 2-3	1 2 3	.....	Slightly irritating	Dry throat	None
58	.....	.....	.....	.....	.....	None
59	3 2 1	1 3 2	.....	Least irritating	Bad taste	None
60	1-3 2	1 2 3	.....	.....	Dry nose	None
61	.....	1 3 2	Increased secretion	Burning	.....	None
62	1-3 2	1 3 2	.....	.....	Bad taste	None
63	1 2 3	1 2 3	Least irritating	Most irritating	.....	None
64	3 1-2	3 1-2	.....	Least irritating	.....	None
65	.....	.....	.....	.....	.....	None
66	3 2 1	3 2 1	.....	Least irritating	.....	None
67	2 1-3	2 1-3	Cough, rough throat	Cough, rough throat	Cough, rough throat	2 least
68	1-3 2	1-3 2	.....	.....	Clearing throat, sting	3 most
69	1-3 2	1 3 2	Less dryness	Less dryness	Most irritating	None
70	1 2 3	1 2 3	Least irritating	Most irritating	Slightly irritating	2 least 1 most
71	1 2-3	1 2-3	Least irritating	Hoarseness (cold)	Irritating to nose	3 most
72	1-2 3	2 1 3	.....	Harsh	.....	2 most
73	1 3 2	1-3 2	.....	.....	.....	None
74	2 3 1	2 3 1	Hyperemia of throat	Slightly irritating	.....	1 most
75	3 2 1	.....	Irritating to throat	.....	Slightly dry nose	None
76	2 1-3	1 2 3	Irritating to tongue	Irritating to tongue	Least irritating	3 most
77	3 2 1	3 2 1	.....	.....	Slightly burning tongue	None
78	1 2-3	.....	Not as harsh	Strong taste	No symptoms	None
79	1-3 2	1 3 2	.....	.....	.....	None
80	1 3 2	1 2 3	.....	Less dryness	Dry throat, secretion	None
81	.....	.....	.....	.....	.....	None
82	1-2 3	1-2 3	.....	Most irritating	.....	None
83	1-3 2	1-3 2	.....	.....	Most irritating	None
84	1-3 2	.....	Irritating to throat	Dry throat	Dry throat	None
85	.....	3 1-2	Less dryness	Dry throat	Less dryness	1 least
86	2 3 1	2 1 3	More irritating than 2	Slight dryness	Less irritating	2 most
87	.....	1 2 3	.....	.....	.....	1 most
88	1-3 2	1-3 2	Good	Good	Irritating	None
89	3 1-2	3 2 1	.....	Cough	Irritating	None
90	3 2 1	3 2 1	.....	Best	Better than 1	None
91	1 2-3	1 2 3	.....	Irritating to tongue	Slightly irritating	None
92	.....	.....	.....	.....	.....	None
93	1-3 2	1 3 2	Slight cough	.....	Distasteful	None
94	1-3 2	3 2 1	.....	.....	Irritating	2 most
95	.....	1 2 3	.....	.....	.....	1 most
96	1 2-3	1 2-3	.....	Occasionally irritating to throat	Dry throat	None
97	2 1-3	2 1-3	Husky voice	Clearing throat	Less clear, bad flavor	None
98	.....	.....	Stronger	.....	.....	None
			(D) Subjects for whom the number of cigarets smoked per day was not specified			
99	1-2 3	1-2 3	.....	Dry throat	.....	None
100	.....	.....	.....	Increased secretion	.....	None
101	.....	2 1-3	.....	.....	.....	None
102	.....	2 1-3	.....	.....	.....	None

* All subjects continued to smoke approximately the same number of cigarets they had used before the test.

† A dash between the number indicates no preference.

SUBJECTIVE REPORTS

In connection with the second phase of the investigation, if consideration is given only to the subjects' reports of adverse symptoms believed by them to have resulted from irritation caused by smoking cigarets from the various lots and indefinite answers, such as those concerning likes or dislikes, flavor or "good" or "bad," are eliminated, the reports are found to be so evenly divided as to have no significance (23 each). However, all subjective reactions of any type are reported in table 3.

LOCAL EFFECTS OF CIGARET SMOKE

The local effects of cigaret smoke on the mouth, throat and nose are debatable. I am unable to pick out a "smoker's throat." It is questionable whether redness of the mucosa indicates irritation from smoking. Excessive serous secretion or salivation of short duration may be an

TABLE 4.—*Lymphoid Hyperplasia of Pharynx and Number of Cigarets Smoked Daily*

No. of Cigarets per Day	Number of Subjects	Percentage with Lymphoid Hyperplasia
5 to 14	41	37
15 to 24	132	48
25 to 40	48	42
Average for group	224	44

indication. The subjective sensation of dryness or burning does not seem to have an associated objective manifestation. The cough, especially in the morning, appears to be due to the mucus secreted by the lung and bronchial mucosa in an effort to remove the nonabsorable constituents of the inspired smoke. The hygroscopic agent would seem to have little or no effect on this secretion.

The possibility of a relation existing between the amount of smoking and the lymphatic hyperplasia of the pharynx present in some persons was considered. A total of 224 subjects were examined with this object in view. Varying amounts of hyperplasia of the lymphatic follicles or bands of the pharynx were observed in 44 per cent. As shown in table 4, enlargement of the lymphatic tissue was found in 37 per cent, of the group of 41 subjects who smoked from five to fourteen cigarets per day, a percentage slightly smaller than the average for the group as a whole. This enlargement was observed in 48 per cent of the group of 132 subjects who smoked from fifteen to twenty-four cigarets per day, a percentage somewhat higher than the average. However, in the group of 51 subjects who smoked from twenty-five to forty cigarets per day the incidence of lymphatic hyperplasia was 42 per cent, slightly smaller

than the average. As a result it is concluded from the tabulation of this group that no definite relationship exists between the enlargement of the lymphatic tissue of the pharynx and the number of cigarets smoked per day.

As in the preceding investigation,[6] the controls or untreated cigarets were favored less than the two other types. This finding may be accounted for by the increased burning rate of dry tobacco, which produces a higher temperature of the smoke. This is in accord with the belief stated by Titkemeyer[7] that the deleterious effects on the buccal mucous membrane are due partly to the raising of the temperature in the mouth and that this factor is more important than the chemical constituents of the tobacco. The observations made in the present investigation would seem to support this view.

CONCLUSIONS

Careful objective examination failed to show any significant difference in irritation of the mucosa of the nose or throat by cigarets moistened with glycerin, those moistened with diethylene glycol or those with no hygroscopic agent.

The subjective symptoms or sensations of irritation, when present, were not marked enough in respect to distribution, character or degree to justify definite conclusions.

Lymphoid hyperplasia of the pharynx does not appear to have any relation to the number of cigarets smoked.

7. Titkemeyer, W.: Die wirksamen Substanzen des Tabakrauches und deren Resorption durch den menschlichen Körper, Med. Welt **11**:209 (Feb. 13) 1937.

THE GALVANIC REACTION IN GUINEA PIGS

I. THE NORMAL GALVANIC REACTION

A. R. BUCHANAN, M.D.
AND
LAURA D. LADD, B.A.
UNIVERSITY, MISS.

The reactions of experimental animals and of human beings to stimulation with the galvanic current have been investigated since early in the nineteenth century. Not only the reactions of normal subjects, but the reactions of animals and human beings with all types of experimentally produced and spontaneous lesions have been the subject of numerous investigations. Experiments on the latter group were carried out in an attempt to establish galvanic stimulation as a clinical procedure which might replace, or at least serve as an adjunct to, performance of the caloric and the rotation test for vestibular function.

Nystagmus, postural reactions, such as turning and rotation of the head, subjective vertigo and a tendency to fall have all been observed as a result of galvanic stimulation of the ears. The nystagmus has received more attention than the other reactions, because of an attempt of the investigators to correlate the galvanic reaction with the reactions elicited by caloric and rotatory stimulation. The direction of the postural reaction in normal subjects has been established as toward the anode; i. e., if the anode was in contact with the right ear, the head was turned toward that side and rotated so that the right ear was lowered. There was also a tendency for the subject to fall to the right.

Ritter [1] was apparently the first author to investigate the effects of galvanic current in the production of vertigo. He was particularly interested in the amounts of current necessary to elicit responses of various kinds in persons of different age and sex. He placed the electrodes in various places—on the forehead, under the eyes, on the zygomatic processes and under the ears. He frequently observed a definite vertigo on the application of the current and expressed the opinion that this was evidence that the entire brain was affected by the current.

From the Department of Anatomy, University of Mississippi, and the Institute of Neurology, Northwestern University Medical School.

1. Ritter: Ueber die Anwendung der Voltaischen Säule, J. d. pract. Heilk. **17**:30, 1803.

Purkinje [2] described the effects of the flow of the galvanic current through the skull in man. He observed the tendency to fall toward the anode and the subjective sensation of vertigo. The sensation was upward from the right to the left if the copper (positive) electrode was in the right ear and the zinc (negative) electrode in the left ear and reversed if the position of the electrodes was reversed. He also assumed that the galvanic falling and the vertigo were due to an effect of the current on the brain.

Hitzig [3] published the results of his experiments on animals and human beings. He observed the tendency to fall toward the side of the anode and the subjective sensation of vertigo, or the sensation of objects in the room rolling toward the side of the cathode. He observed, further, a deviation of the eyes toward the anode. Like Ritter and Purkinje, he explained the reaction as due to cerebral irritation.

Breuer [4] stimulated one labyrinth, placing the other electrode on the anterior abdominal wall. He expressed the conviction that the galvanic reaction was due not to stimulation of the brain but to stimulation of the semicircular canals.

Mackenzie [5] described the two most commonly used methods of producing galvanic vertigo and nystagmus. The first method consisted of applying the two electrodes to the opposite sides of the head, the positive on one side and the negative on the other. By this method a positive reaction could be produced in normal subjects with a current of 2 to 4 milliamperes. The reaction consisted of rotatory nystagmus to the side of the cathode, a sensation of falling in the sagittal plane toward the side of the anode and, finally, actual falling to the anodal side. By this method, according to Mackenzie, the two sides were irritated simultaneously, and it was impossible in cases of pathologic imbalance to tell exactly which side was pathologically underirritable or overirritable.

The second method consisted of applying one electrode to the region of the ear and the other to some distant point. The wider the separation of the electrodes, the greater was the resistance and, naturally, the stronger the current necessary to produce the reaction. Bárány found it necessary to use 10 to 12 milliamperes to produce a marked reaction by this method; Mackenzie, however, produced visible reactions with 4 to 8 milliamperes.

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2. Purkinje, J.: Ueber die physiologische Bedeutung des Schwindels und die Beziehung desselben zu den neuesten Versuchen über die Hirnfunctionen, Mag. f. d. ges. Heilk. **23**:284, 1827.

3. Hitzig, E.: Ueber die beim Galvanisieren des Kopfes entstehenden Störungen der Muskelinnervation und der Vorstellungen vom Verhalten im Raume, Arch. f. Anat., Physiol. u. wissensch. Med. **37**:716, 1871.

4. Breuer, J.: Ueber die Function der Bogengänge des Ohrlabyrinthes, Med. Jahrb. **4**:72, 1874.

5. Mackenzie, G. W.: Labyrinthine Papers, Philadelphia, 1913.

Jensen [6] found that among normal pigeons the reaction to galvanic current of one and the same subject was not exactly the same from time to time. He found also that after prolonged stimulation larger amounts of current became necessary to produce the response. In the normal bird a reaction could usually be produced with a slight amount of current (0.05 milliampere).

Marx [7] reported that a current of 0.2 to 0.4 milliampere was sufficient to cause deviation of the eyes of normal guinea pigs toward the anode (head fixed). A current of 0.5 to 1 milliampere produced a nystagmus in the opposite direction.

Babinski,[8] in attempting to exclude one ear, placed one electrode in the opposite ear and the other 0.5 cm. anterior to it on the same side of the head of a pigeon with exposed labyrinth. He found that the head was inclined to the stimulated side when the electrode in the ear was positive; when it was negative the head was inclined to the opposite side somewhat less actively, a finding essentially in agreement with that of Spamer.[9] Babinski concluded that the vestibule and the ampullae were more essential than the semicircular canals in producing the galvanic effect, on the basis of experiments involving destruction of one or the other structure. In a paper written later in the same year [10] he noted, in addition to the inclination of the head, a rotation of the neck toward the anode. He described as the point of election for stimulation in the guinea pig an area just above the tragus which was most sensitive to either positive or negative stimulation. He was able to obtain inclination and rotation of the head with the electrode in this position with less than 1 milliampere of current.

Brünings [11] disputed the anelectronic and catelectronic hypothesis of Bárány, which explained the galvanic effect as due to a lessening or heightening of the irritability and conductivity of the vestibular nerve by the flow of the current, on the hypothesis that by such increase or decrease in the irritability of the nerve the transmission of the tonus-regulating impulses from the labyrinth was either facilitated or hindered.

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6. Jensen, P.: Ueber den galvanischen Schwindel, Arch. f. d. ges. Physiol. **64**:182, 1896.

7. Marx, H.: Ueber den galvanischen Nystagmus, Ztschr. f. Ohrenh. **63**:201, 1911.

8. Babinski, J.: Sur le mécanisme du vertige voltaïque, Compt. rend. Soc. de biol. **55**:350, 1903.

9. Spamer, C.: Experimenteller und kritischer Beitrag zur Physiologie der halbkreisförmigen Kanäle, Arch. f. d. ges. Physiol. **21**:479, 1880.

10. Babinski, J.: Sur les mouvements d'inclination et de rotation de la tête dans le vertige voltaïque, Compt. rend. Soc. de biol. **55**:513, 1903.

11. Brünings: Ueber neue Gesichtspunkte in der Diagnostik des Bogengangapparates, Verhandl. d. deutsch. otol. Gesellsch. **19**:192, 1910; Beiträge zur Theorie, Methodik und Klinik der kalorimetrischen Funktionsprüfung des Bogengangapparates, Ztschr. f. Ohrenh. **63**:20, 1911.

Brünings maintained that such electrotonus as is produced in other nerves could not exist in the vestibular nerve, which is deeply embedded in the temporal bone. To prove his point he stimulated the peripheral labyrinth calorically and by rotation and determined that the effects of these types of stimulation were not increased by simultaneous stimulation of the same ear negatively with the galvanic current. Similarly, the effect was not decreased by placing the positive electrode on that ear. Brünings then hypothesized that the galvanic reaction depends on an electrical transmission (cataphoresis) by means of the fine particules in the endolymph, or on a transmission through the fluid itself (electroendosmosis) such as one can obtain through certain fluid mediums in capillary tubes. This would bring about an adequate stimulation of the sensory epithelium through a mechanical displacement of the structure on which the epithelium rests, just as one elicits a reaction by rotation or caloric stimulation. Thus did he explain why a constant current was necessary and why the direction of the nystagmus elicited was dependent on the direction of the current, since the movement of the suspended particles in a fluid medium brought about by an electric current depends on the direction of the current and changes with it.

Uffenorde,[12] working with apes, found, like many other authors, that the galvanic reaction could still be elicited after complete labyrinthectomy. He held that Brünings' theory of cataphoresis was untenable. Like Jensen,[6] he found that there were great individual variations in the amount of current necessary for the elicitation of galvanic nystagmus and that the amount of current necessary was not constant for the two ears in a single animal.

Fischer and Wodak [13] compared the effects of galvanic stimulation with those obtained by caloric stimulation. They found that irrigation of the ear with cold water produced bodily reflexes (such as turning of the head) toward the stimulated side, while irrigation with warm water produced the same reflexes toward the opposite (unstimulated) side. In galvanization the anode had the same effect as the cold water and the cathode the same as the warm water.

Pollak [14] conducted experiments on normal and deaf-mute persons and found that normal human beings, without exception, showed on the

12. Uffenorde, W.: Zur Bewertung der Augenmuskelreaktionen bei Labyrinthreizung und der Reaktionen bei elektrischen Kleinhirnreizungen nach experimentellen Untersuchungen am Affen, München. med. Wchnschr. **59**:1213 and 1277, 1912.

13. Fischer, M. H., and Wodak, E.: Beiträge zur Physiologie des menschlichen Vestibularapparates: I. Die "vestibularen Körperreflexe" und die "Fallreaktion," Arch. f. d. ges. Physiol. **202**:523, 1924.

14. Pollak, J.: Ueber den galvanischen Schwindel bei Taubstummen, Arch. f. d. ges. Physiol. **54**:188, 1893.

application of the galvanic current to the ear typical movements of the head and the eyes, which were absent in 30 per cent of deaf-mutes. He expressed the belief that the origin of the reaction must, therefore, be in the vestibular apparatus.

Malassez[15] also examined deaf-mutes and concluded that the integrity of the semicircular canals was essential for postrotatory nystagmus, but that the galvanic reaction could be obtained if a part, at least, of the vestibular nerve was still intact. He assumed that the galvanic reaction, characterized by an inclination of the head and body with associated vertigo, is a manifestation of a disturbance of the vertical sense, a sense which, according to André Broca, is localized in the saccular and utricular maculas. The pressure exerted by the endolymph on the maculas, a pressure varying with the position of the head, is supposed to inform the subject as to his vertical direction. The galvanic current, by exciting inequally the utricular and saccular rami of either ear, would then provoke a disturbance of the sense of vertical position, which accounts for the reactions observed.

Mygind,[16] conducting vestibular examinations on normal persons and on some with traumatic vestibular lesions, found that in the former group galvanic nystagmus could usually be obtained with 3 milliamperes of current but might require 10 to 13 milliamperes, the average being 5 milliamperes. The figures varied greatly from one person to another, from time to time in the same person and in the same subject during a single examination. In the latter series he found the galvanic falling reaction, which was highly recommended by Rhese[17] in cases of trauma, to be unreliable, not allowing any conclusion to be drawn either as to the localization of the disease or as to its organic or functional nature. The galvanic falling reactions were examined when the current was broken while the subject was sitting in a chair. As a rule, in normal subjects there was a sense of falling in the direction opposite to the nystagmus; however, even in the normal persons, paradoxic reactions were observed.

Bard,[18] as have many other authors, observed that when one electrode was in contact with the ear and the other on the back of the neck the nystagmus produced by positive stimulation of the ear was like that

15. Malassez, J.: Excitation mécanique et électrique du vestibule chez les sourds-muets, Compt. rend. Soc. de biol. **89**:1256, 1923.

16. Mygind, S. H.: Traumatic Vestibular Diseases, Acta oto-laryng. **1**:515, 1918; Vestibular Examinations in Normal Individuals, ibid. **1**:527, 1918.

17. Rhese, H.: Ueber die traumatische Läsion der Vestibularisbahn, insbesondere über den Sitz der Läsion, Ztschr. f. Ohrenh. **70**:262, 1914.

18. Bard, L.: Du mécanisme et de la signification du nystagmus voltaïque, Ann. de méd. **5**:239, 1918.

from irrigation with cold water; when the negative electrode was in contact with the ear, the nystagmus was like that from irrigation with warm water.

Mann [19] reasoned that, if under the influence of the galvanic current the body lowers toward one side, there must have occurred a change in tonus or in the innervation of the muscles on that side. He found, indeed, that if two objects of equal weight were held in the outstretched hands and a galvanic current passed through the ear, the object in the hand on the side toward the anode seemed heavier, which substantiated his view that there was a decrease in muscular tonus on that side.

Blonder [20] described a clinical method for "testing the integrity of the vestibular arc" by observing the falling reaction produced by a galvanic current when the subject stood on a balance board which consisted of a platform 16 inches (41 cm.) wide, 21 inches (53 cm.) long and ¾ inch (2 cm.) thick, under which was a fulcrum 3½ inches (9 cm.) wide and ⅞ inch (2.2 cm.) thick. The patient was placed in the normal standing posture on the balance board. Circular electrodes covered with moist cloth were placed, one over the mastoid process on one or the other side, and the second on the sternum. Closure of the circuit produced lowering of the platform on the anodal side. Usually 0.5 to 2 milliamperes of current was sufficient to produce the reaction in normal persons; rarely as much as 5 milliamperes was required. Patients with "both labyrinths dead" failed to react to galvanic stimulation by this method when as much as 20 milliamperes was used.

The literature contains diverse and conflicting views as to the value and the accuracy of the reaction to stimulation with the galvanic current. Some investigators, notably Babinski, Rhese and Blonder, have considered the galvanic falling reaction, or at least the postural reaction, a valuable asset in the diagnosis of vestibular disease. Others, such as Mygind and Uffenorde, have found the reaction in normal persons so variable that they have stated that it is unreliable for clinical diagnosis.

The point of attack, or *Angriffspunkt,* of the galvanic current, through which it acts in producing a reaction, has engaged the attention of many workers. In order to utilize the galvanic reaction in the diagnosis of disease, the structures which bring about the reaction must, of course, be known. Such has been the object of our research; however, before one can draw conclusions concerning the reactions under pathologic conditions, it is necessary to analyze carefully the reaction in normal animals. The present report deals with the reaction elicited in a series of normal guinea pigs.

19. Mann, L.: Ueber die galvanische Vestibularreaktion, Neurol. Centralbl. **31**:1356, 1912.

20. Blonder, E. J.: Galvanic Falling in Clinical Use, Arch. Neurol. & Psychiat. **37**:137 (Jan.) 1937.

MATERIALS AND METHODS

For the original determinations of the galvanic reactions a small box was built which would comfortably accommodate a guinea pig. The box was balanced on a narrow fulcrum in such a way that a slight shift in the center of gravity of the animal tipped it to right or left, the principle of this apparatus being the same as that of the balance board described by Blonder.[20] One electrode was attached to the dorsal surface of the neck and the other to the tragus by means of skin clips and gauze soaked with physiologic solution of sodium chloride. The prongs of the skin clips pierced both the gauze and the skin and conducted the current to the subcutaneous tissues. When the electrodes were properly attached, the animal was placed in the balance box and the cover placed on the box. The wires from the electrodes were led out of the box through holes in the cover and were attached to the galvanic machine; this instrument was equipped with a rheostat which permitted one to start with a milliampere reading of zero and increase the amount of current gradually to as high as 20 milliamperes. There was also a switch which permitted reversal of the current without change of the electrodes or of the connections of the wires to the poles of the machine. It was possible, then, to subject the ear under observation to positive (anodal) or negative (cathodal) stimulation at will.

When the normal animals were tested with this apparatus, a current of 1 to 3 milliamperes was sufficient to cause tipping of the box toward the anode. Thus, if the right ear was positively stimulated, the animal's center of gravity was shifted to the right enough to cause the box to tip in that direction; if the right ear was negatively stimulated, the box tipped to the left.

Although the percentage of paradoxic reactions (tipping in the wrong direction) was only 4.2, the method was criticized on the basis that, since the box was so delicately balanced, its tipping to one or the other side might be due to painful stimulation, and reactions in the proper direction might be largely coincidental. In order to overcome this criticism, the apparatus was considerably modified.

The modification consisted in mounting the box used in the previous setup on ball bearings; a counterbalance was then rigidly suspended from the bottom of the box. This counterbalance was heavy enough to maintain the box, with the guinea pig in place, in the vertical position. A short lever, on the extremity of which a stylus was fixed, was attached to the box; thus, by the use of a kymograph a graphic record of the animal's reactions to the galvanic current could be obtained. The animal was tied in place in the box by means of a cord attached to each foot and led out through properly placed holes in the box; thus the cover of the box could be discarded and the actual postural reaction of the animal more adequately studied and compared with the direction of the tipping of the box. A photograph of the apparatus is reproduced (fig. 1). Since the stylus was placed at the right side of the animal, a reaction to the right was recorded as a downward excursion of the stylus on the kymograph; a reaction to the left produced an upward excursion. Reactions to the right, or downward excursions, could be produced by positive stimulation of the right ear or negative stimulation of the left ear. Reactions could be produced in normal guinea pigs with currents varying from 2 to 5 milliamperes.

This apparatus obviated the necessity for righting the box after each stimulation, since the counterbalance did this automatically. Sometimes, however, if the animal did not return completely to its original position, the base line from which the next reaction was recorded was moved slightly upward or downward (fig. 2 *C*). Usually, however, breaking of the current caused a sufficient reaction in the

opposite direction to reestablish the original base line. The possibility which was suggested in connection with the original apparatus, that reactions were obtained in the proper direction by balancing the box in such a way that there was a mechancial advantage in that direction, did not exist with this setup.

RESULTS

Kymographic records of the galvanic reaction have been obtained from 30 guinea pigs, which were subsequently operated on. Post-operative records were obtained, which will be discussed in later reports.

Typical records are reproduced (fig. 2). Representative portions of each animal's record were selected, contact negatives were made and

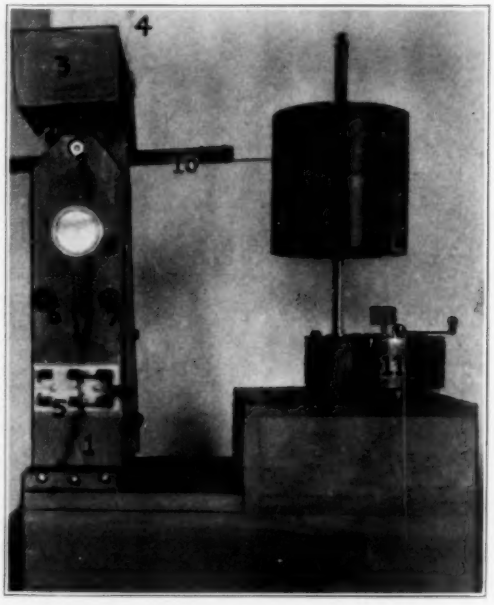

Fig. 1.—Apparatus used for recording galvanic reactions in guinea pigs: (1) rigid upright supporting shaft on which the box is mounted; (2) shaft on which the box is mounted by means of ball bearings; (3) box in which the animal is placed, shown tipped to the right; (4) one of the wires which lead to the electrodes attached to the animal's ears and neck; (5) reversing switch facilitating either positive or negative stimulation of the ear in question; (6) and (7) resistance controls; (8) milliammeter; (9) counterbalance for the box; (10) lever supporting the stylus, and (11) kymograph.

contact positives were printed from the negatives. Examination of the records discloses only one paradoxic reaction (fig. 2 *B*), when positive stimulation of the left ear gave a reaction to the right instead of to the left. Of the two hundred and eighteen separate reactions recorded from 30 animals, eleven, or 5 per cent, were in the wrong direction. All the animals, however, which gave paradoxic reactions gave some reac-

tions in the proper direction. The animal represented in figure 2 *B* and others which gave reactions in the wrong direction seemed more sensitive to the current than those which gave constant and proper reactions; hence pain was probably an important factor in their behavior. When the animal tended to struggle on the application of the current, the direction of the reaction recorded on the kymograph was not indicative of a true galvanic reaction.

Direct observation of the animal's behavior on application of the current has added nothing to the classic descriptions by Babinski and by other older workers. The head is turned and rotated toward the side of the anode, and there is an apparent increase in tonus of the limbs on

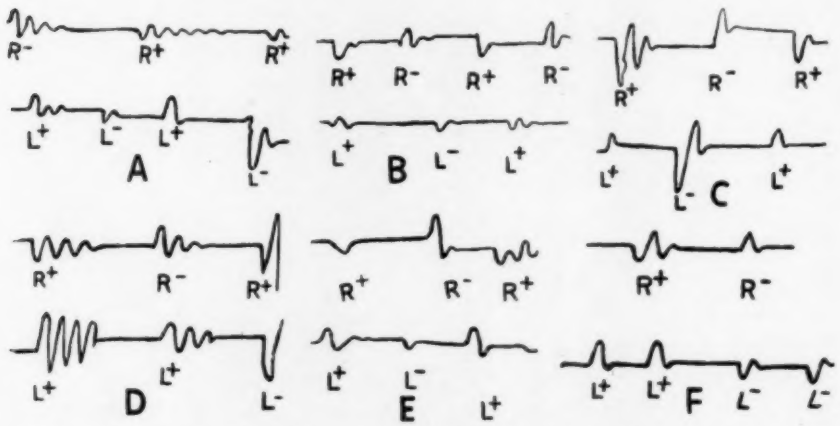

Fig. 2.—Records of the galvanic reactions of 6 normal guinea pigs. R^+ indicates anodal stimulation of the right ear; R^-, cathodal stimulation of the right ear; L^+, anodal stimulation of the left ear, and L^-, cathodal stimulation of the left ear.

the side of the cathode, combined with some decrease on the side of the anode, which accounts for a slight shift in the center of gravity toward the anodal side. Nystagmus always appears, which is directed (quick component) toward the cathode.

It can readily be seen from the records presented that the strength of the reaction, as represented by the amount of excursion of the stylus, was not constant for the animal, for the ear being studied or for the sign of the electrode. Some animals, for example that represented in figure 2 *F*, gave uniform responses on positive or negative stimulation of either ear. Others, like that represented in figure 2 *C*, were erratic in the strength of their responses. There is no evidence, so far as the small amounts of current used are concerned, that either negative or positive stimulation is the more effective. However, if an animal is placed on the floor, with electrodes connected in the usual way, nega-

tive stimulation seems the more effective when large amounts of current (15 to 20 milliamperes) are used; in such an experiment positive stimulation will cause the animal to lean toward the ear stimulated, while negative stimulation of the same ear results in actual falling toward the opposite side, the animal lying on its side, unable to regain the upright position so long as the current is flowing.

Prolonged repetition of stimuli tends to decrease the reaction, a finding in agreement with that of Jensen.[6] When a point was reached in a series of stimulations at which 5 milliamperes no longer produced a reaction, an increase in the current to 10 milliamperes served to bring about a recurrence.

Since pain apparently tended to disturb the normal galvanic response, it was thought that one might eliminate it by locally anesthetizing the points of application of the electrodes. For this purpose 0.4 cc. of 0.1 per cent solution of nupercaine [21] was injected at the root of the tragus and into the skin on the back of the neck. That the anesthetic was effective was evident on the application of the skin clips, which apparently caused little or no pain.

Local anesthesia at the points of application of the electrodes tended to eliminate irreguluarities in the galvanic response and to make for more uniformity in the force of the reaction. This fact can be ascertained by examination of figure 3, in which records on the same animal are presented before and after injection of nupercaine. The record of the right ear (fig. 3 *A*) showed more improvement than that of the left (fig. 3 *B*). A note made at the time of the experiment stated that anesthesia was apparently more complete in the right than in the left tragus. Since the local anesthesia did eliminate, to some extent, the irregularities in the kymographic record and did not interfere with the normal reaction of the animal, it was made a regular part of the procedure of obtaining records both from the animals which had been submitted to operation and from those on which no operation had been performed. Local anesthetization produced no significant change in the percentage of paradoxic reactions.

Whether the labyrinth is essential to the normal galvanic reaction remains to be proved, and it would seem that this problem could be better studied by a method which utilizes kymographic recording of the reaction. Dohlman [22] concluded that Scarpa's ganglion and the central portion of the eighth nerve were sufficient to produce the galvanic response. Blonder,[20] on the other hand, found that patients with "dead

21. The nupercaine was supplied by Ciba Pharmaceutical Products, Inc., Lafayette Park, Summit, N. J.

22. Dohlman, G.: Experimentelle Untersuchungen über die galvanische Vestibularisreaktion, Acta oto-laryng. (supp.) **8**:1, 1929.

labyrinths" failed to react. It seems probable also that some of the nerves supplying the skin at the usual points of application of the electrodes to the ears may be partially responsible for the transmission ot the galvanic current to the brain centers, which must in turn be responsible for the changes in tonicity of the muscles of the limbs. These would include the fifth, the seventh, the tenth and the eleventh cranial nerve, as well as the upper three cervical nerves. The fibers of the ninth nerve which supply the middle ear may also be a factor in the response.

Local anesthetization of the skin at the points of application of the electrodes has eliminated pain as a factor in the true galvanic reaction. Conduction of the current through the sensory nerves which supply the area, however, is apparently unimpaired by such a procedure. We have studied the effect of local application of nupercaine to the posterior tibial

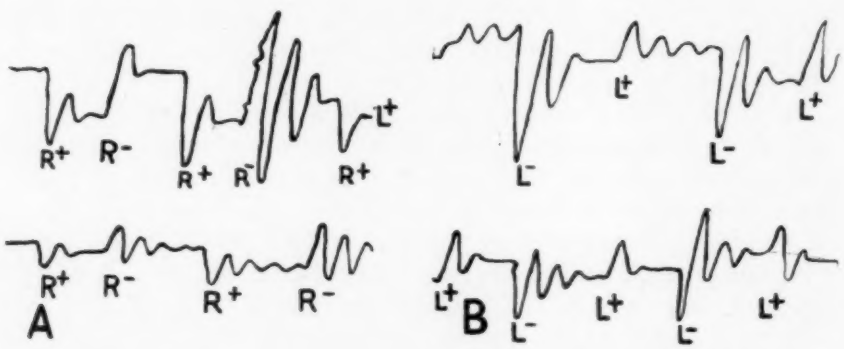

Fig. 3.—Records of the galvanic reactions of a normal animal before (upper line) and after (lower line) anesthetization by means of nupercaine of the areas of application of the electrodes. Reactions from stimulation of the right ear are shown in *A* and those from stimulation of the left ear in *B*.

nerve in the rat. After the nerve had been dissected out, nupercaine in 1 : 1,000 solution was applied to it, either by contact with a cotton pledget soaked in the solution or by injection into the nerve sheath. The effect of faradic stimulation of the nerve was studied at intervals for thirty to forty minutes, and it was found that a reaction could be obtained in the muscles supplied by the nerve, manifested by flexion of the toes, throughout this period, with the secondary coil set at 12 cm. What was more important from our standpoint was that we were able to obtain a similar reaction on the making and the breaking of the galvanic current with only 0.5 to 1 milliampere. The effect of nupercaine on conduction was further studied when the skin on the dorsum of the foot was locally anesthetized. Stimulation applied to the anesthetized area at intervals for thirty minutes, with either the faradic or

the galvanic (make and break) current, elicited a reaction. Uhlmann [23] obtained a considerable reduction in sensitivity to faradic current in the sensory nerves of the frog, but his coil separation was 35 cm., plus angulation of the secondary coil to 70 degrees. He noted also some suppression of the reaction of the gastrocnemius muscle on stimulation of the sciatic nerve.

From our standpoint, however, and as a result of our experiments with the rat, we cannot eliminate conduction over the sensory nerves supplying the external ear as a probable factor in the galvanic reaction.

SUMMARY

The reaction of guinea pigs to galvanic current with one electrode applied to the tragus and the other to the dorsum of the neck has been studied. An apparatus has been devised whereby the reactions may be recorded on a kymograph. The records obtained from a series of 30 animals indicate that the reaction has a definite direction in relation to the sign of the electrode in contact with the ear. Uniformity in the force and direction of the reaction has been facilitated by local anesthesia of the skin at the points of application of the electrodes, which eliminates the factor of pain. There is an apparent variation from animal to animal in sensitivity to the flow of the galvanic current; this has, of course, been found in human beings also.

The direction of the reaction follows the classic descriptions, being always toward the anode and away from the cathode. With the currents used (2 to 5 milliamperes) neither anodal nor cathodal stimulation has proved more effective. The strength of the reaction, however, can be influenced by increasing the amount of current, more forcible reactions being obtained when larger amounts of current are used.

Galvanic nystagmus directed away from the anode was present, but was not studied, our interest being confined to an attempt to duplicate the galvanic falling reaction described and used clinically by Blonder.[20]

Dr. Edwin J. Blonder cooperated in the early part of this work at the Institute of Neurology, Northwestern University Medical School.

23. Uhlmann, F.: La percaïne, un nouvel anesthésique local, Arch. internat. de pharmacodyn. et de thérap. **36**:253, 1929.

THE GALVANIC REACTION IN GUINEA PIGS

II. THE REACTION FOLLOWING LABYRINTHECTOMY

A. R. BUCHANAN, M.D.

AND

LAURA D. LADD, B.A.

UNIVERSITY, MISS.

The literature covering the normal galvanic reaction in animals and in human beings has been reviewed in a previous report;[1] a method of studying the reaction in guinea pigs was described in the same report.

Many investigators have concerned themselves with the question as to whether the galvanic reaction is altered by disease of, or mechanical destruction of, the labyrinth. Ewald[2] carried out research on pigeons after unilateral and bilateral labyrinthectomy. He reported that in an animal with both inner ears completely destroyed high currents caused no turning of the head. In a pigeon that has had unilateral labyrinthectomy, if the anode was placed on the labyrinthectomized side and the cathode on the sound side there ensued turning of the head toward the anode and rotation of the head in the same direction, both of which reactions increased with increase in the strength of the current. If the positions of the electrodes were reversed no turning or rotation of the head resulted, even with high currents. On the basis of these findings he concluded: (1) that the galvanic reaction occurs through stimulation of the inner ear, (2) that stimulation occurs in the ear on which the cathode is placed and (3) that the stimulation of an ear effects a turning of the head and a spiral rotation of the neck toward the nonstimulated side.

Jensen[3] found the difference between normal and labyrinthectomized animals to be in the strength of current required to produce the galvanic reaction, the latter group requiring more current for less movement. He concluded that the normal galvanic reaction is dependent on

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From the Department of Anatomy, University of Mississippi Medical School, and the Institute of Neurology, Northwestern University Medical School.

1. Buchanan, A. R., and Ladd, L. D.: The Galvanic Reaction in Guinea Pigs: I. The Normal Galvanic Reaction, Arch. Otolaryng., this issue, p. 124.

2. Ewald, J. R.: Die Abhängigkeit des galvanischen Schwindels vom inneren Ohr, Centralbl. f. d. med. Wissensch. **28**:753, 1890; Physiologische Untersuchungen über das Endorgan des Nervus octavus, Wiesbaden, J. F. Bergmann, 1892, pp. 227-252.

3. Jensen, P.: Ueber den galvanischen Schwindel, Arch. f. d. ges. Physiol. **64**:182, 1896.

the undisturbed condition of the labyrinths, while the abnormal response obtained in the labyrinthectomized animals is dependent on direct stimulation of motor nerve centers.

Beyer and Lewandowsky [4] investigated the galvanic reaction in dogs, cats and an ape. They performed unilateral and bilateral labyrinthectomy and compared the results of galvanic stimulation in labyrinthectomized and in normal animals. After unilateral extirpation of the labyrinth, the galvanic reaction was normal, except that turning of the head toward the labyrinthectomized side with the anode placed on that side required less current than did turning of the head in the other direction when the anode was on the sound side. With the cathode on the labyrinthectomized side a stronger current was required to produce a response than when it was on the sound side. In animals subjected to bilateral labyrinthectomy these investigators obtained exactly the same reaction to galvanic stimulation as in normal animals, except that a stronger current was required. They assumed that the galvanic reaction in these cases came about through stimulation of the central nervous system or, specifically, of the vestibular nuclei in the medulla.

Marx [5] found that after complete destruction of the labyrinths the galvanic reaction was still normal. He expressed the opinion that conclusions as to the condition of the labyrinths could not be drawn from galvanic investigation and that differential diagnosis of diseases of the nerve apparatus and the auditory organs is impossible by this method.

Blau,[6] working with cats, guinea pigs and an ape, found that stronger currents were required to elicit nystagmus if the labyrinth was partially destroyed; however, when it was completely destroyed, nystagmus could still be elicited with the galvanic current. He examined the temporal bones microscopically and pointed out as significant the fact that the cells of Scarpa's ganglion and the eighth nerve were still intact in the labyrinthectomized animals.

Dohlman [7] investigated the galvanic reactions in animals with the eighth nerve sectioned or with the vestibular ganglion removed and in animals with lesions in the vestibular nuclei. He concluded that the galvanic reaction is of no significance in the diagnosis of the functional condition of the labyrinths or the sensory epithelium and that the reaction was elicited by direct stimulation of the cells of the vestibular ganglion, a conclusion in agreement with that of Blau.

4. Beyer, H., and Lewandowsky, M.: Experimentelle Untersuchungen am Vestibularapparat von Säugetieren, Arch. f. Anat. u. Physiol. (Phys. Abt.), 1906, p. 451.

5. Marx, H.: Ueber den galvanischen Nystagmus, Ztschr. f. Ohrenh. **63**: 201, 1911.

6. Blau: Experimentelle Studien über den galvanischen Nystagmus, Ztschr. f. Ohrenh. (supp.) **69**:6, 1913.

7. Dohlman, G.: Experimentelle Untersuchungen über die galvanische Vestibularisreaktion, Acta oto-laryng. (supp.) **8**:1, 1929.

Babinski, Vincent and Barré [8] performed unilateral and bilateral labyrinthectomy on guinea pigs. After unilateral destruction of the labyrinth conjugate deviation of the head and eyes was wanting or diminished when the positive electrode was on the side of the lesion; with the current reversed (the anode on the uninjured ear), there was conjugate deviation, equal to or greater than normal to that side. The current employed varied from 5 to 10 milliamperes. In bilaterally labyrinthectomized animals conjugate deviation of the head and eyes toward the anode was still present; with large amounts of current the animal fell to the anodal side or lay without moving ("cataleptic state").

Strehl [9] conducted experiments on frogs and pigeons and was unable to determine any difference in the galvanic reaction before and after destruction of the labyrinths.

Babinski [10] concluded from observations on patients that when unilateral disease of the auditory apparatus or of the eight nerve exists the tendency is for the head to be inclined toward the side of the lesion, regardless of the position of the anode (whether on the sound or the diseased side). When galvanic stimulation was applied in cases of bilateral lesions the tendency was for the head to be inclined toward the side which was more severely involved.

Bárány [11] investigated the galvanic reaction but was unable to verify Babinski's findings as to the tendency to inclination of the head toward the diseased side in aural disease. He attached no significance to the galvanic reaction, since he could observe no parallelism between the reaction and the results of other clinical tests and no correlation with the pathologicoanatomic picture.

Rhese [12] stated that he was able to make a differential diagnosis between peripheral and central lesions of the vestibular mechanism by means of the galvanic reaction. He found that the galvanic reaction is usually normal when peripheral lesions are present. In cases in which galvanic nystagmus could not be elicited, a galvanic falling reaction, usually in the direction of the expected nystagmus, was still manifested on the opening of the circuit, whereas, in general, in traumatic

8. Babinski, J.; Vincent, C., and Barré, A.: Vertige voltaïque: Perturbation dans les mouvements des globes oculaires à suite de lésions labyrinthiques experimentales, Rev. neurol. **25**:253, 1913.

9. Strehl, H.: Beiträge zur Physiologie des inneren Ohres, Arch. f. d. ges. Physiol. **61**:205, 1895.

10. Babinski, J.: De l'influence des lésions de l'appareil auditif sur le vertige voltaïque, Compt. rend. Soc. de biol. **53**:77, 1901.

11. Bárány, R.: Untersuchungen über den vom Vestibularapparat des Ohres reflektorisch ausgelösten rhythmischen Nystagmus und seine Begleiterscheinungen, Monatschr. f. Ohrenh. **40**:193, 1906.

12. Rhese, H.: Ueber die traumatische Läsion der Vestibularisbahn, insbesondere über den Sitz der Läsion, Ztschr. f. Ohrenh. **70**:262, 1914.

lesions of the vestibular mechanism the falling occurs in the direction of the slow component. He found, also, like Babinski, that the most frequent variation in the falling reaction is a fall toward the injured side, regardless of which ear is stimulated.

Mann [13] elicited the galvanic falling reaction in animals with complete unilateral destruction of the labyrinth but observed that the strength of current required on the labyrinthectomized side to elicit turning and rotation of the head was greater than that required on the sound side. He concluded that in cases in which the galvanic reaction is lost not only must the end organ be destroyed but the vestibular nerve must have undergone degeneration.

To Blonder [14] belongs the credit for devising a method for utilizing the galvanic falling reaction as a clinical test of the "integrity of the vestibular arc." The patient stands on the platform of a so-called balance board; one electrode is placed over the mastoid and the other over the sternum. Closure of the circuit produces lowering of the board on the anodal side. Blonder has reported that patients with both labyrinths dead fail to react to galvanic stimulation when as high as 20 milliamperes are used.

The object of the present study has been to determine the effect of unilateral and bilateral labyrinthectomy on the galvanic reaction of guinea pigs as determined by the method described in a previous report,[1] which method simulates that used by Blonder [14] with human beings.

PROCEDURE

The galvanic reactions of the first 5 animals included in the present report were examined by means of a simple balance box, or a small box delicately balanced on a narrow fulcrum. With the animal in the box and electrodes attached to one or the other ear and the dorsum of the neck, the strength of current required to cause a shift in the animal's center of gravity sufficient to tip the box to one or the other side was recorded.

The procedure with the other animals has consisted of: (1) preoperative examination of the galvanic reaction with the aid of our modified apparatus consisting of a box large enough to accommodate the animal comfortably, mounted on ball bearings and counterbalanced from below, which provided a kymographic record of the animal's reaction; (2) unilateral labyrinthectomy; (3) observations of the postoperative behavior of the animal, and (4) postoperative examination of the galvanic reaction. Caloric stimulation of the ears was also carried out before and after operation, since this method of testing the integrity of the labyrinth is in general use and has been clinically proved. In 3 animals the remaining labyrinth was subsequently destroyed, and records of the galvanic reaction were obtained.

The labyrinthectomy was a relatively simple operation. The auditory bulla was exposed by an incision caudal to the external ear and an opening made through

13. Mann, L.: Ueber die galvanische Vestibularreaktion, Neurol. Centralbl. **31**:1356, 1912.

14. Blonder, E. J.: Galvanic Falling in Clinical Use, Arch. Neurol. & Psychiat. **37**:137 (Jan.) 1937.

the bulla, exposing to view the medial wall of the middle ear. The vestibule, semicircular canals and cochlea were then destroyed by means of a dental drill. Deviation of the eyes toward the side of the destruction and spontaneous nystagmus to the opposite side appeared during the course of the drilling.

The postoperative behavior of the animals differed in no particular from the classic description by Magnus,[15] including the already mentioned deviation of the eyes and spontaneous nystagmus, the rolling toward the side of the lesion and the turning of the head, the spiral rotation of the head and thorax and the concavity of the vertebral column, all toward the side of the labyrinthectomy. Compensation occurred gradually, and after two to three weeks only deviation of the eyes and turning and rotation of the head remained.

All the animals were eventually killed, and the temporal bones were fixed, sectioned and stained for microscopic study. The degree of destruction of the internal ear varied somewhat but was practically complete in all cases included in this report—certainly complete enough to compare with such spontaneous lesions of the internal ear as occur in human subjects.

TABLE 1.—*Results of Galvanic and Caloric Stimulation in 5 Animals in Which Labyrinthectomy Had Been Done on the Right* *

Animal number	9	10	25	26	27
Anode in right ear	R7	R2	R12	R8	R3
Anode in left ear	L5	L2	L2.5	L3	L3
Caloric stimulation in right ear	—	—	+	—	—
Caloric stimulation in left ear	++	++	++	++	++

* The following abbreviations are used: R indicates tipping of the box to the right; L, tipping to the left; the numbers following R and L, the number of milliamperes of current required to produce the reaction; —, the absence of caloric nystagmus; ++, normal caloric nystagmus, and +, a reduced response to caloric stimulation.

Nupercaine (1:1,000 solution) was injected at the site of application of the electrodes in all the more recently studied animals, in order to eliminate any extraneous movement attributable to pain caused by the current; the use and rationale of this local anesthesia were discussed in the previous report.[1]

RESULTS

The results of galvanic stimulation in the first 5 animals, in each of which labyrinthectomy had been produced on the right side only, are summarized in table 1 and compared with the results of caloric stimulation. The animals listed are the ones which were examined by means of the simple balance box. Only positive stimulation of the ears was carried out; all subsequent animals were given both positive (anodal) and negative (cathodal) stimulation of both ears. The figures indicating the strength of current required to produce tipping of the box to the right or the left represent the averages arrived at after several determinations on each animal.

With animals 10 and 27, tipping occurred as readily when the anode was applied to the right as to the left or sound ear, although the response

15. Magnus, R.: Körperstellung, Berlin, Julius Springer, 1924.

to caloric stimulation was lost in the right ear and was still present in the left. Animals 9 and 26 required current of greater strength on the right or labyrinthectomized side, a finding in agreement with those of Jensen,[3] Beyer and Lewandowsky,[4] Mann [13] and others. It was necessary with animal 25 to employ a current of 12 milliamperes before tipping of the box to the right occurred, although a slight nystagmus could be produced by caloric stimulation of the right ear, indicating that labyrinthine destruction was not complete.

TABLE 2.—*Results of Galvanic Stimulation in 11 Animals Before and After Labyrinthectomy* *

		Number of Paradoxic Reactions per Ten Stimuli						Amount of Current (Milliamperes)						Sign of More Effective Current					
				Postoperative Record						Average of Postoperative Record						Postoperative Record			
	Side of Unilateral Labyrinthectomy	Preoperative Record		Unilateral		Bilateral		Preoperative Record		Unilateral		Bilateral		Preoperative Record		Unilateral		Bilateral	
Animal No.		R	L	Op.	No Op.	R	L	R	L	Op.	No Op.	R	L	R	L	Op.	No Op.	R	L
92	Left	0	0	0	0	....	....	2½	2½	5	2½	..	..	0	0	0	0	..	..
94	Left	0	0	0	0	....	....	2½	2½	7	2½	..	..	—	—	0	—	..	..
96	Left	0	0	0	1.2	0	0	2½	2½	5	2½	4	5	0	0	—	+	—	—
100	Right	0	1.9	0	2.8	....	....	2½	5	7½	4	..	..	0	0	—	0	..	..
106	Left	0	0	1.8	0	0	0	2½	2½	6	2½	5	5	0	—	—	—	—	—
107	Left	0	2.8	0	5.0	....	....	2½	2½	5	2½	..	..	0	0	+	—	..	..
108	Left	0	0	0	10.0	....	....	2½	2½	20	20	..	..	+	+	—	+	..	..
109	Left	0	0	0	0	....	....	2½	2½	8	6	..	..	0	+	—	0	..	..
110	Right	0	0	0.6	1.7	5.0	5.0	5	5	10	5½	6	4	0	—	—	—	0	0
111	Left	0	0	0.8	3.3	....	....	5	7	7	4	..	..	0	0	—	0	..	..
112	Left	1.2	0.8	5.0	0.2	....	....	2	2	7	2½	..	..	0	—	—	0	..	..
Average.....		0.109	0.55	0.704	2.2	1.6	1.6	2.9	3	7.9	4.8	5	4.6						
Percentage..		1.09	5.5	7.04	22.0	16.6	16.6												

* The following abbreviations are used: R indicates the electrode placed in the right ear; L the electrode placed in the left ear; Op., the electrode placed in the ear operated on and No Op, the electrode placed in the normal ear. Under the heading "Sign of More Effective Current," — indicates that the negative current was the more effective; +, that the positive current was the more effective, and 0 that the positive and the negative current were equally effective in eliciting a response.

Obviously, the results obtained from this small preliminary group of animals were not conclusive, although 3 of the 5 did require current of greater strength for a response from the labyrinthectomized side. This is significant in the light of the average strength of current required to produce reactions in the animals, recorded in table 2.

The second group included 11 animals, on 3 of which studies were conducted after bilateral labyrinthectomy. The results of galvanic stimulation following unilateral and bilateral lesions are summarized in table 2, and are compared with the preoperative records. The postoperative figures summarize the records obtained at intervals of three to five days during the postoperative course of each animal.

Paradoxic Responses.—For the statistical study of the paradoxic reactions all the records on a single animal were examined; the total

number of stimuli were ascertained, together with the total number of paradoxic reactions, and the number of the latter per ten stimuli was then recorded. From these figures, the percentage of paradoxic responses, indicated at the bottom of table 2, were readily arrived at. In all the reactions recorded preoperatively, the percentage of paradoxic responses from the right ear was 1.09, while that from the left was 5.5 All the paradoxic responses were accounted for by 3 animals (table 2); the remaining 8 animals showed no preoperative reactions in the wrong direction. After unilateral labyrinthectomy the paradoxic responses on the labyrinthectomized side amounted to 7.04 per cent and those on the sound side to 22 per cent.

Of the 3 animals in which bilateral labyrinthectomy was performed, only 1 showed paradoxic reactions, which were equal in percentage of occurrence on the two sides. Since, however, paradoxic reactions occurred so frequently in this animal (110), the percentage of such reactions in our small group of bilaterally labyrinthectomized animals amounted to 16.6 for either ear.

Animal 108 gave 100 per cent paradoxic reactions on the sound side, a figure considerably above the mean. Autopsy showed that the posterior wall of the middle ear had been perforated, with destruction of a small area on the lateral surface of the pons. The presence of this central lesion may have accounted for the different results obtained from this animal.

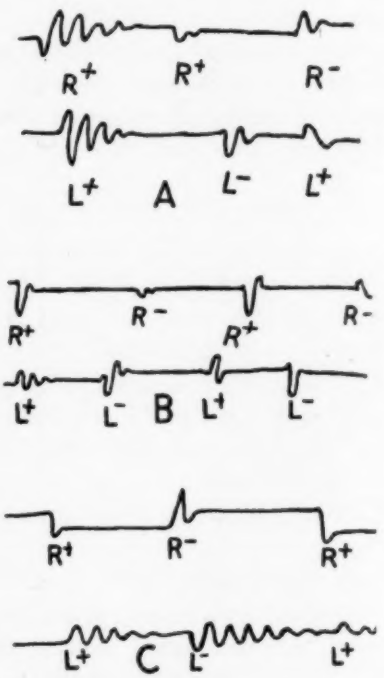
We have attempted to correlate the paradoxic reactions in our guinea pigs with Babinski's observations but have been unable to do so. We have found the number of paradoxic reactions away from the side of the lesion to be approximately equal to the number toward that side.

Amount of Current Required.—The average amount of current necessary to produce a reaction in this group of guinea pigs preoperatively was 2.9 milliamperes on the right side and 3 milliamperes on the left. After unilateral labyrinthectomy an average strength of 4.8 milliamperes was required on the sound side and of 7.9 milliamperes on the labyrinthectomized side.

After bilateral labyrinthectomy the amounts of current necessary to produce reactions were essentially equal on the two sides, being 5 milliamperes on the right and 4.6 milliamperes on the left. These amounts were somewhat higher than those required preoperatively but were less than those required on the labyrinthectomized side after unilateral operation. These findings substantiate those of Beyer and Lewandowsky [4] but do not agree with the observations of Blonder,[14] who obtained no reactions in patients suffering from bilateral loss of the labyrinth.

Sign of the More Effective Current.—Preoperative records indicate that negative and positive stimulation of the ears are about equally effective in eliciting the galvanic reaction. After unilateral labyrinthectomy negative stimulation was more effective in 72 per cent of the cases when the labyrinthectomized side was stimulated. The sound side remained as before operation. In a few instances the positive current was the more effective, but the reactions were in the same proportion before and after operation.

Bilaterally labyrinthectomized animals also showed a tendency to greater response to the negative current. Of these animals, cathodal



A, typical record of the galvanic reaction in a normal animal. *B*, record of the galvanic reaction ten days after labyrinthectomy on the left. *C*, record of the galvanic reaction after the destruction of the right labyrinth in the same animal. R^+ indicates anodal stimulation of the right ear; R^-, cathodal stimulation of the right ear; L^+, anodal stimulation of the left ear, and L^-, cathodal stimulation of the left ear. Anodal stimulation of the right ear and cathodal stimulation of the left ear are normally recorded as downward excursions of the stylus; cathodal stimulation of the right ear and anodal stimulation of the left ear are normally recorded as upward excursions of the stylus.

stimulation produced a more active response in 2, while cathodal and anodal stimulation were equally effective in 1.

Caloric Stimulation.—After labyrinthectomy nystagmus was not elicited by caloric stimulation of the labyrinthectomized ear or ears of

any of the animals in this group. On the basis of this recognized test of labyrinthine function, then, the operations carried out did eliminate the labyrinth from consideration in the galvanic reactions recorded post-operatively.

Kymographic Records.—Kymographic records of the galvanic reactions of animal 96 before operation, after destruction of the left labyrinth and after subsequent destruction of the right labyrinth are presented in the figure. This animal gave no paradoxic reactions in either ear before operation. After destruction of the left labyrinth paradoxic reactions occurred in the ratio of 1.2 to 10 when the sound ear was stimulated. One such reaction can be seen in the figure (B, R^-). Also, it was necessary to use twice as much current to produce a reaction in the labyrinthectomized side as in the sound side (table 2). After destruction of the right labyrinth current of greater strength was necessary to produce a reaction in either ear than before the first operation, but there were no paradoxic reactions.

SUMMARY AND CONCLUSIONS

The reactions of 16 guinea pigs to the flow of galvanic current have been studied before and after labyrinthectomy. Unfortunately, as was noted in the first study in this series,[1] a small percentage of normal guinea pigs react in the wrong direction to the application of galvanic current, the number of paradoxic reactions ranging as high as 2.8 per ten stimuli in 1 animal in this series. Since, however, certain changes have been noted in the reaction of labyrinthectomized animals, it seems essential that such changes should be recorded if the galvanic reaction is to be considered as an adjunct to the caloric and other tests for labyrinthine function.

As has been noted, the stimulation of the ears of normal subjects results in a postural reaction toward the side of the anode, which shifts the center of gravity of the animal to that side. With the animal placed in a balance box, this shift in center of gravity is sufficient to tip the box toward that side. By recording the tipping of the box and the sign and strength of stimulation on a kymograph, we have been able to arrive at some rather definite conclusions as to the effect of labyrinthectomy on the galvanic reaction.

Unilateral labyrinthectomy in guinea pigs predisposed to reactions in the wrong direction. It is of especial interest that such paradoxic reactions were approximately three times more frequent on the sound side than on the side of the labyrinthectomy.

Paradoxic responses in bilaterally labyrinthectomized animals occurred less frequently than in those with only one labyrinth destroyed. Two such animals gave no reaction in the wrong direction.

Reactions in the wrong direction were more frequently away from the side of the lesion than toward it, a circumstance which is not in accord with Babinski's observations.[10]

Unilaterally labyrinthectomized animals required current of greater strength on both the labyrinthectomized and the sound side to produce a reaction than did normal animals; however, considerably more was required on the labyrinthectomized side than on the sound side.

Bilaterally labyrinthectomized animals required current of greater strength to produce reactions than did normal animals, but the amount required was approximately equal on the two sides and was less than that required on the side of the labyrinthectomy in unilaterally labyrinthectomized animals.

Anodal and cathodal stimulation were equally effective before operation; after either unilateral or bilateral labyrinthectomy, however, cathodal stimulation seemed to be the more effective.

Case Reports

FUNGOUS INFECTIONS OF THE SINUSES

H. D. Rentschler, M.D., and C. H. Scheifley, M.D., Sayre, Pa.

Fungi are frequent saprophytic invaders of the human body. Occasionally they become parasitic and produce definite pathologic processes. Almost every organ in the body has been invaded by pathogenic fungi, and in this respect the accessory nasal sinuses are no exception. Although the condition is rare, enough cases of mycotic infection of the nasal sinuses have been reported to establish it as a definite clinical entity.

The earliest cases on record are those of Hernu [1] and Plaignaud,[2] who in 1791 made the diagnosis of fungous infection of the maxillary sinus from the gross appearance of the disease.

Zarniko [3] in 1891 reported the first case of aspergillosis of the sinuses. The chief symptoms were those of chronic suppurative sinusitis with occlusion of the nose. The organism was determined by microscopic examination to be Aspergillus fumigatus.

In 1893 Mackenzie [4] described a case of aspergillus invasion of the maxillary sinus. The symptoms were chronic nasal discharge, swelling of the right side of the face and over the root of the nose and pain over the right eye and ear. Microscopic sections showed aspergilli, which were believed to be of the fumigatus type.

A case of suppuration of the maxillary sinus due to A. fumigatus was described by Harmer [5] in 1913. The patient had persistent discharge of mucus from the nose, containing an occasional thick, membranous brownish yellow cast, which looked like wet blotting paper. A

From the Section of Eye, Ear, Nose and Throat, Guthrie Clinic.

1. Hernu, J. J.: Fongus du sinus maxillaire, abandonné à luimême; ses ravages; la mort qui les suivit; dissection, J. de chir. **2**:278, 1791.

2. Plaignaud: Observation sur un fongus du sinus maxillaire, J. de chir. **1**:111, 1791; J. de méd. **87**:244, 1791.

3. Zarniko, C.: Aspergillusmykose der Kieferhohle, Deutsche med. Wchnschr. **17**:1222, 1891.

4. Mackenzie, J. H.: Preliminary Report on Aspergillus Mycosis of the Antrum Maxillaire, Bull. Johns Hopkins Hosp. **4**:9, 1893.

5. Harmer, D.: Suppuration of the Antrum Due to Aspergillus Fumigatus, J. Laryng., Rhin. & Otol. **28**:494, 1913.

cure resulted from the use of potassium iodide internally after lavage of the sinus had failed. The diagnosis in this case was confirmed by microscopic examination and culture.

Tilley [6] in 1915 reported 5 cases of aspergillosis of the maxillary sinus. The chief symptoms were nasal obstruction, sneezing, mucopurulent discharge, occasional expulsion of small masses of whitish gray, semitranslucent viscous material and neuralgic pains in the cheek, face and eye. A Caldwell-Luc operation resulted in cure in all 5 cases. The diagnosis in these instances was based on the microscopic appearance. Skillern [7] found material that resembled cottage cheese in the washings from a maxillary sinus. He identified aspergilli, probably of the species fumigatus, from this material.

More recently Adams [8] described an instance of infection of the maxillary, ethmoid and sphenoid sinuses, in which A. fumigatus was recovered from these parts. The patient was a Negress, aged 32, who for six years had had a chronic discharge and attacks of bleeding from the left side of the nose, obstruction to breathing, frequent colds in the head, protrusion and redness of the left eye and headaches in the left frontal, orbital and temporal regions. Her history revealed an attack of arthritis involving all the joints, which had occurred one year before admission. Biopsy material from the ethmoid region showed dense fibrous tissue with mycelial threads and giant cells. Culture identified the fungus as A. fumigatus. Radical operation showed the left sinuses to be filled with this tumor-like material. The removal of this tissue, combined with the internal use of large doses of potassium iodide, finally resulted in a cure.

Stevenson [9] in 1933 reported a case of chronic sinusitis in which a fungus was recovered and cultured from the washings of both maxillary sinuses. Later he [10] reported 7 similar cases, in which diagnosis was based on the isolation of fungous forms from antral washings. Culture and microscopic study showed an organism resembling the cells of the human body so closely that it was difficult to differentiate it. He placed the fungus in the class Phycomycetes and the order Mucoraceae

6. Tilley, H.: Aspergillosis of the Nasal Accessory Sinuses, J. Laryng., Rhin. & Otol. **30**:145, 1915.

7. Skillern, R. H.: Aspergillosis of the Maxillary Sinus, Laryngoscope **31**: 946, 1921.

8. Adams, N. F., Jr.: Infection Involving the Ethmoid, Maxillary, and Sphenoid Sinuses and the Orbit Due to Aspergillus Fumigatus: Report of Case, Arch. Surg. **26**:999 (June) 1933.

9. Stevenson, H. N.: Mucormycosis of the Maxillary Sinus, Arch. Otolaryng. **18**:775 (Dec.) 1933.

10. Stevenson, H. N.: Mycotic Infections of the Maxillary Sinuses, Arch. Otolaryng. **20**:340 (Sept.) 1934.

and proposed for it the name Mucorhistoides. Clinically and in gross pathologic picture the condition differed in no way from other chronic infections of the sinuses. Treatment was that employed against the usual sinusitis. The author suggested that a constant state of infection with fungi would easily explain the type of sinal infection which produces almost constant slight nasal catarrah with acute exacerbations from time to time.

Kelly [11] in a recent paper stressed that aspergillus infection of the nose and its accessory sinuses is not so rare as the scanty references in the textbooks would lead one to believe. His report is based on 7 cases. Aspergilli were observed in the nose in 4 and in the maxillary sinus in 3. The most important diagnostic sign, he stated, was the discharge from the affected side of pieces of whitish membrane or lumps of tough, gelatin-like green substance at intervals varying from a day to several weeks. For treatment he recommended the Caldwell-Luc operation.

Actinomycosis of the accessory nasal sinuses is rare. Stevenson and Adair Dighton [12] in 1911 reported a case of actinomycotic osteomyelitis of the sphenoid sinus with intracranial invasion and death. In 1923 New and Figi [13] described 2 cases of actinomycosis of the nasopharynx which spread to the paranasal sinuses. Kramer and Som [14] cited a case of primary actinomycotic infection of the wall of the sphenoid sinus with the formation of abscess of the brain and a fatal outcome.

REPORT OF CASE

The patient, a housewife aged 49, was first seen on Feb. 8, 1937, when she was admitted to the Robert Packer Hospital, with the diagnoses of infectious arthritis and hyperthyroidism, both of one year's duration. There was a history of a chronic nasal discharge from the left side and some obstruction to breathing of several months' duration. The patient noticed a few mild pains on the right side of the face. Aside from influenza in 1918, she had had no severe illnesses.

Examination gave the following results: The tonsils were moderately enlarged and showed slight chronic inflammation. The right nasal fossa showed no abnormalities. On the left, the space between the lateral wall and the middle turbinate body was filled with a semi-inspissated, nonodorous material. It was difficult to see up under the vault beneath the middle turbinate body. The material had the appearance of wet blotting paper and came away in chunks,

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11. Kelly, A. B.: Aspergillosis of the Nose and Maxillary Antrum, J. Laryng. & Otol. **49**:821, 1934.

12. Stevenson, E., and Adair Dighton, C. A.: Actinomycotic Meningitis Due to Sphenoid Suppuration Causing Death, Ophthalmoscope **9**:402, 1911.

13. New, G. B., and Figi, F. A.: Actinomycosis of the Head and Neck, Surg., Gynec. & Obst. **37**:617, 1923.

14. Kramer, R., and Som, M. L.: Actinomycosis of the Sphenoid with Actinomycotic Meningitis and Brain Abscess, Ann. Otol., Rhin. & Laryng. **44**: 973, 1935.

leaving slight bleeding areas. Small black particles were recovered as this substance was removed. Roentgenograms revealed distinct opacity of the left maxillary sinus.

Smears were taken from the substance in the left nasal fossa. They showed spore forms and mycelial processes. On culture a dark-colored growth was obtained, which was identified as Aspergillus. It was classified as the species niger, because of the black particles which were found in the fungous material from the nose. A diagnosis of infection of the left maxillary sinus due to Aspergillus niger was made.

On March 9 a turbinectomy on the left side and a window resection of the intranasal wall of the left maxillary sinus were done. After three weeks of treatment by antral lavages, the patient was symptom free, and the antrum was clear.

The patient was next seen on March 9, 1938. At this time there were no symptoms; the sinuses were clear and the nose normal. There had been no improvement in the arthritis.

COMMENT

Whalen [15] stated in a discussion of pathogenic fungi that only by the routine examination of the washings from the antrum and the discharges from the other sinuses would a possible fungous infection be detected. However, chronic sinusitis with nasal obstruction, the presence of dark-colored solid masses in the discharge and finally the discovery of a fungoid mass protruding into the nasal cavity are sufficient for a presumptive diagnosis. In the case reported, this last sign led to a search for the organism.

It is interesting to note that in Adams's case, in one of Stevenson's cases and in the case here reported, arthritis complicated the picture.

The discovery of fungi in pathologic material is best made microscopically with an alcoholic solution of sodium sulfide, which clears the specimens of blood, fibrin and pus cells.[16] The organisms are easily cultured on Sabouraud's or Shaw's medium. A. fumigatus assumes a greenish tinge at first but after ten days takes on a smoky appearance. A niger soon shows the surface of the medium covered with black granules. A light Gram's stain will aid in the examination and classification of the fungi.

Fungous infections of the sinuses do not always respond to simple lavage. In Zarniko's case great improvement resulted from washings, but this treatment was supplemented by potassium iodide in daily doses of from 2 to 4 Gm. (30 to 60 grains). Mackenzie resorted to an antral window but reported improvement only. Simple lavage proved ineffective in Harmer's case. A cure was effected in ten days when he used sodium iodide in rapidly increasing doses, starting with 0.13 Gm. (2 grains) daily. Tilley used the Caldwell-Luc operation and obtained

15. Whalen, E. J.: Pathogenic Fungi, Arch. Otolaryng. **24**:436 (Oct.) 1936.

16. Cornbleet, T.: A Reagent for Demonstrating Fungi in Skin Scrapings and Hair, J. A. M. A. **95**:1743 (Dec. 6) 1930.

cures. Adams failed to effect a cure even with radical surgical intervention, and it was not until huge doses of potassium iodide were employed, from 6.5 to 13 Gm. (100 to 200 grains), that a cure was effected.

As far as can be determined the case here reported is the only one of aspergillosis of the maxillary sinus cured by a simple intranasal window operation. The patient had received potassium iodide in the form of compound solution of iodine while in the hospital in preparation for a thyroidectomy. This amounted to only 0.3 Gm. (5 grains) daily for twenty days. Larger doses were not given because of the existing hyperthyroidism. There was no improvement in the patient's nasal symptoms during this period.

Just as with the usual acute and chronic sinusitis, the method of treatment must be adjusted to the demands of the individual case. In addition to the standard methods of attack, the use of potassium iodide is a measure of proved value.

OSTEOCHONDROMA OF THE ESOPHAGUS REMOVED PERORALLY WITH THE AID OF A SPECULUM

With Review of the Benign Tumors of the Esophagus

ERNEST REEVES, M.D., PASSAIC, N. J.

It is always with hesitation that one reports any sort of medical rarity, lest it be proved later that inadequate search of the literature has caused failure to discover that the supposed curiosity has been previously observed.

Nevertheless, I should like to report an instance of osteochondroma in the esophagus of a 74 year old man.

Benign tumors of the esophagus are rare. E. Paterson in 1932, after careful search of the literature, found only 61 cases. According to Chevalier Jackson, who certainly has the most experience with and knowledge of diseases of the esophagus, "The case records of the bronchoscopy clinics of Pittsburgh and Philadelphia show histories of the following benign tumors in which the esophageal walls were involved: granuloma, edematous polypi, papilloma, keloid, fibroma, adenoma, lipoma, myoma, myxoma, angioma, varix, hematoma, and cystic growths." More than half of these growths were polypi, papillomas and leiomyomas.

Many of the benign tumors are pedunculated as a result of the pulling action of deglutition. Cases have been reported in which the pedicle was so long as to permit the appearance of the tumor in the mouth.

The polypi are usually smooth mucous hyperplasias covered by epithelium. They nearly always hang on a long pedicle.

Papillomas are really papillary fibromas. They often are vascular and therefore bleed easily.

Adenomas are rare and often lobulated.

Lipomas are found chiefly at autopsy as small nodules in the submucosa. Sometimes, however, they grow large and become pedunculated.

Leiomyomas are usually found in the anterior wall at the lower level of the esophagus, for the musculature of the upper third is of the striped variety. They are small and usually do not cause any symptoms at all; but if they grow or are annular they may cause considerable stenosis.

The cysts, according to Kaufmann, may be classified into four groups: (1) small multiple retention cysts of the ducts of mucous glands, occurring especially in the upper part of the esophagus; (2) small cysts starting from the islands of gastric mucosa; (3) follicular or cystic esophagitis when the relatively few mucous glands participate in chronic esophagitis, and (4) ciliated epithelial cysts (according to Wyss and Zahn) originating from embryonic structures snared off while the epithelium was still ciliated and cylindric.

Trespe found cartilage in the walls of some of these cysts.

The symptoms of benign tumor of the esophagus are usually those of more or less complete obstruction. A large growth causes difficulty in swallowing, regurgitation and occasionally nausea. The compression of the trachea or the bronchi may cause pulmonary complications, or these may be secondary to the overflow and inspiration of infected secretions. Coughing and choking on taking food are noticed in the latter class of cases.

In making the diagnosis roentgenologic study is helpful. It may clear up difficulties in cases in which the tumor, although benign, invades the esophagus from the outside, pushing the normal esophageal tissues in front of itself. It may also show the location of the tumor and the amount of obstruction it causes.

The sovereign diagnostic method is that of esophagoscopy. Esophagoscopic appearance may show the absence of infiltration and the absence of invasion of normal structures. Benign growths are usually covered by an uninterrupted epithelial surface.

Erosion occurs only after some injury or irritation has occurred. The removal of the growth in whole or in part by means of the esophagoscope enables the physician to make an accurate histologic diagnosis.

None of the authors cited mentioned osteochondromas of the esophagus. This, however, was the type of tumor I removed from the esophagus of P. J. on July 27, 1937.

REPORT OF CASE

P. J., a man aged 74 and weighing 140 pounds (63.5 Kg.), complained of difficulty in swallowing. He was able to swallow; even a large bolus of food could go down with the aid of some water, but it seemed to stop in his chest and often caused considerable pain. As time went on, liquid foods continued to be swallowed easily enough; but the solid bolus of foods caused so much suffering that he was afraid of taking his meals. Consequently he lost a considerable amount of weight—10 to 15 pounds (4.5 to 6.8 Kg.).

These difficulties began about eight months prior to operation and grew progressively worse.

Except for the usual colds the patient was never ill. There was no tuberculosis in his family, and he denied having syphilis. His children and grandchildren were living and healthy. All his life he had had difficulty in breathing through his left nostril. In the past three or four years he had been hawking considerable amounts of brownish yellow pus from his throat on arising.

The nasal septum was deviated to the left and a considerable amount of dried hard grayish yellow crust and pus was found in the nasal passages. This pus could be seen tapering down from the nasopharynx to the oropharynx. The mucous membranes of the nose and throat showed all the signs of chronic inflammation.

The larynx did not show any pathologic conditions and the pyriform sinuses did not show the jacksonian sign—they did not contain any saliva.

Esophagoscopy with a standard 9 mm. esophagoscope showed a tumor on the posterior wall of the esophagus in its midline. It was about 20 cm. from the incisor teeth, somewhat lower than the beginning of the thoracic esophagus, which opened well on inspiration. The wall around the tumor did not appear to be infiltrated and was mobile.

The growth seemed elastic. It could easily be bent by the push of an instrument but would immediately resume its normal position when the force was removed. In appearance it resembled the tip of the little finger penetrating the posterior wall of the esophagus, forming a sharp angle at its base with the esophageal wall.

The tumor was about 11 mm. high and 10 mm. in diameter and was covered with normal mucous membrane which did not show any infiltration, ulceration or redness; and neither did the mucosa of the neighboring part of the esophagus, which was pale and easily movable.

The esophagoscope was replaced by a Jackson esophageal speculum through which the tumor and its surrounding esophagus was anesthetized with a 20 per cent cocaine solution. Then, with cutting forceps, as large a single piece of tumor as possible was removed.

The greater part of the tumor came away with the first bite of the forceps; the remainder was carefully removed piecemeal. No attempt was made at radical removal of the base of the tumor; for normally the esophageal wall is thin, measuring 3 to 5 mm., and is easily perforated. Perforation is often a fatal complication.

Notwithstanding the anesthesia and the previous administration of a full dose of morphine, the operation was painful. For about ten days after the operation the patient had considerable difficulty in swallowing. Discomfort persisted for two months and then disappeared.

At present, six months after the operation, the patient has no difficulty at all in swallowing any kind of food. In January 1938 esophagoscopy and roentgenoscopy did not reveal any kind of esophageal obstruction. The patient has gained 20 pounds (9.1 Kg.), weighing now 160 (72.6 Kg.), and is able to do his regular daily work.

The portion of the tumor removed by the first bite of the cutting forceps was different from anything I expected. It consisted of reddish tissue covered by smooth mucous membrane on its esophageal surfaces.

In the middle of the basilar cut surface, loosely embedded in it and freely movable in all directions, was a distinctly different formation. It looked like half a globe or a cup 5 mm. in diameter and 2.5 mm. deep. The wall of this cup was much harder than any other part of the specimen. It was so firm and elastic as to suggest the presence of cartilage in its wall.

A perusal of Kaufmann's and later of Aschoff's Pathology gave me the impression that the tumor was one of the cysts previously

described, because in the walls of some of these cysts Trespe found cartilage.

The result of the histologic examination performed by Dr. Kastler of St. Mary's Hospital, Passaic, N. J., and that of a later similar examination by Dr. A. A. Eggstone of the Manhattan Eye, Ear and Throat Hospital, New York city, did not confirm my first impression. Dr. Eggstone's description follows:

> The specimen consists of an irregular mass of grayish white tissue measuring 10 x 10 x 5 mm. One portion is rather soft. There is one caplike area of tissue, rather dense and resistant, which measures 5 mm.
>
> Microscopic section reveals stratified squamous epithelial mucosa. The submucosa consists of some smooth muscle with considerable fat and fibrous tissue which is continuous with a rather dense hyaline fibrous tissue zone showing some fibroblastic areas.
>
> This tissue forms the caplike structure above described. Near the concavity the connective tissue is dense and of perichondrial character. In the concavity is a small area of fibrohyaline cartilage with some areas of calcification. There is very little inflammatory reaction present.
>
> Pathologic diagnosis: Esophagus, subesophageal tissues, perichondrium with an osteochondromatous area.

As osteochondroma of the esophagus was not described in the textbooks of pathology quoted and seemingly has not occurred in bronchoscopic clinics in Philadelphia and Pittsburgh, it will be worth while to present photomicrographs of the sections showing the most important observations. These are especially interesting because some of the microscopic slides reveal not only the character of the tumor but also the part of the esophagus in which the tumor was found.

For orientation I shall briefly review the normal histologic anatomy of the esophagus. It has three coats, an external or muscular coat, a middle or areolar coat and an internal or mucous coat.

The surface of the mucous coat is studded with minute papillas and is covered throughout with a thick layer of pavement epithelium. Beneath the mucous membrane, between it and the areolar coat, is a layer of longitudinally arranged smooth muscle fibers. This is the muscularis mucosa. At the commencement of the gullet it is absent or is represented only by a few scattered bundles. Lower down it forms a considerable stratum.

The muscular coat is composed of two planes of fibers of considerable thickness, an external longitudinal and an internal circular plane. The muscle fibers in the upper part of the esophagus are red and consist chiefly of the striped or voluntary variety, whereas below, in the lower part of the esophagus, they consist entirely of the involuntary smooth variety.

The areolar coat connects loosely the mucous and muscular coats.

The esophageal glands are small compound racemose glands scattered throughout the tube. They are lodged in the submucous tissue and open on the surface of the esophagus by long excretory ducts. At

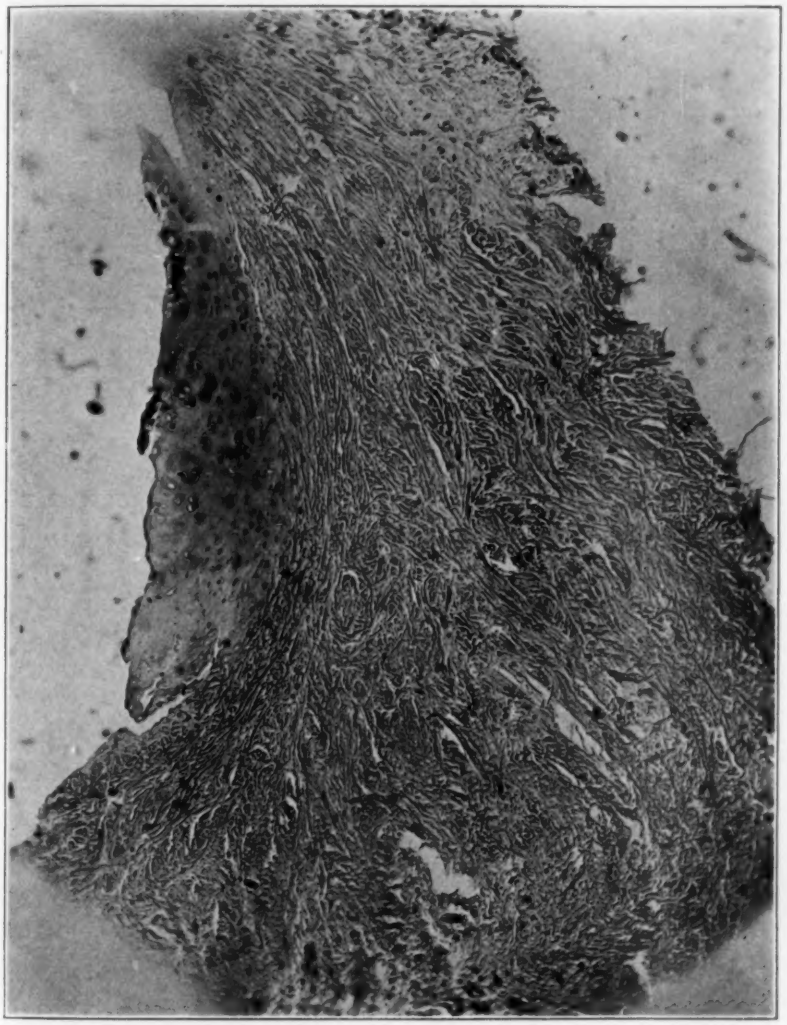


Fig. 1.—Photomicrograph showing the hard caplike structure, with fibrohyaline cartilage and some areas of calcification. Around it the connective tissue is dense and is perichrondrial in character.

the upper part of the tube they are not numerous. At the lower part they form a ring around the cardiac orifice.

The three photomicrographs shown here were taken in the Manhattan Eye, Ear and Throat Hospital and are enlargements of different parts of the same microscopic section.

The scarcity of the glands and the absence of the muscularis mucosa show that the microscopic section is from the upper third of the

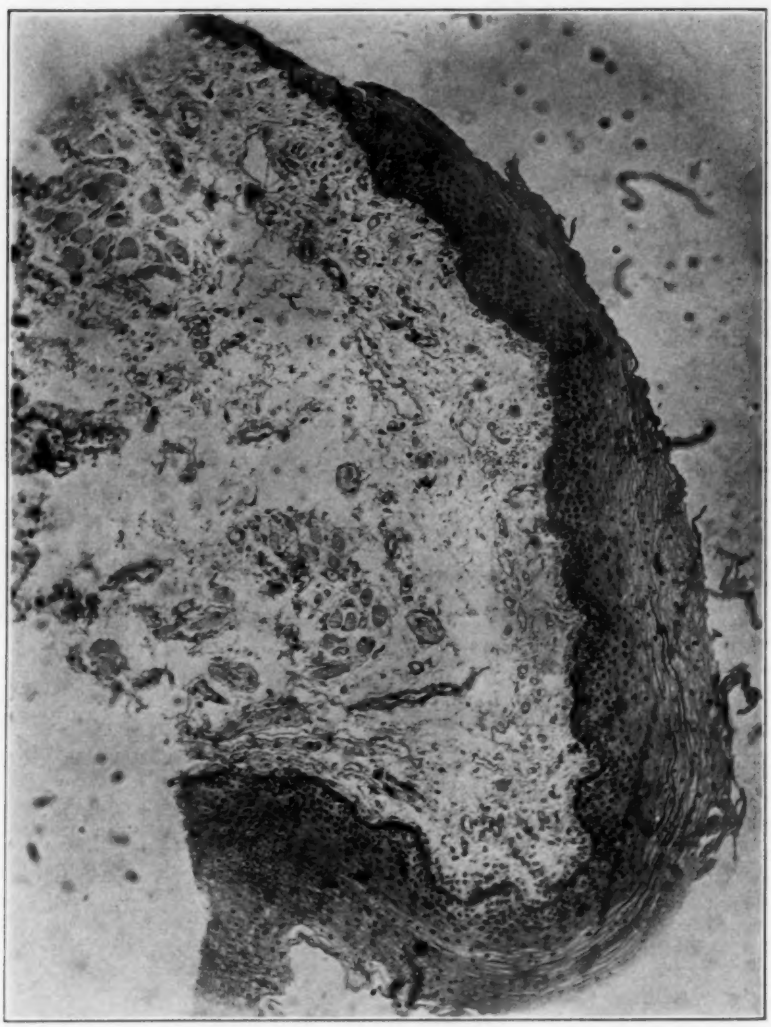


Fig. 2.—Photomicrograph showing the stratified squamous epithelium with almost no glands and no muscularis mucosa.

esophagus. The presence of equal amounts of smooth and striated muscle fibers narrows the location of the growth to the level of the first or second dorsal vertebra.

The origin of the tumor can only be surmised. Dr. Eggstone suggested that it may have originated from the intervertebral disk between the first and second vertebrae, slowly growing into the posterior wall and pushing all the layers of the esophagus in front of itself.

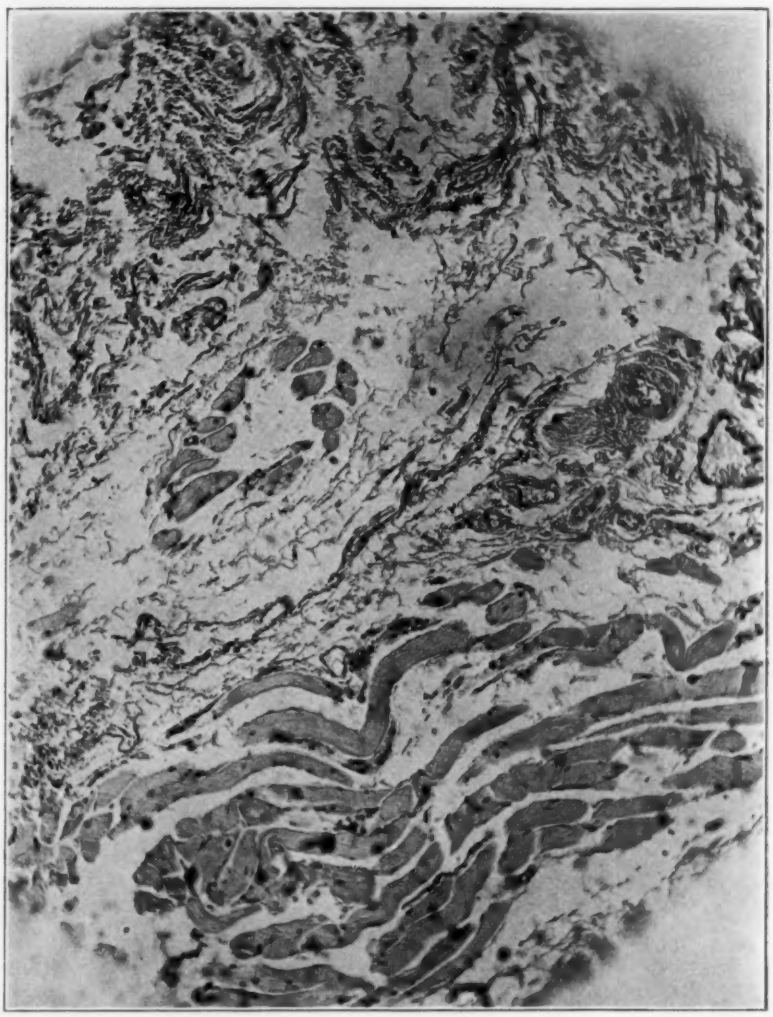


Fig. 3.—Photomicrograph of the muscular coat, showing clearly that it consists of smooth and striated muscle in nearly equal amounts.

To eliminate the obstructing tumor all the esophageal strata had to be removed. Adhesions, however, around the tumor prevented the infection of the mediastinum.

A thorough roentgenologic examination of the vertebrae, performed by Dr. Jahn of St. Mary's Hospital, did not show any exostosis, chondroma or disfiguration of the vertebral column in its upper part. Of course this is a very difficult region for roentgenologic examination, as the scapulas, the clavicles, and the ribs all throw their shadows on the upper part of the thoracic vertebral column.

SUMMARY

An osteochondromatous tumor was found and removed with the aid of an esophageal speculum from the posterior wall of the upper thoracic esophagus 20 cm. from the incisor teeth.

OTITIC MENINGITIS SUCCESSFULLY TREATED WITH SULFANILAMIDE

L. L. White, M.D., Brooklyn

In recent years the literature has recorded and many physicians have observed remarkable recoveries from intensive infection with Streptococcus haemolyticus. A great deal of credit for these cures has been attributed to sulfanilamide, and justly so. In this report, a case of positive meningitis caused by Str. haemolyticus superimposed on acute mastoiditis due to Str. haemolyticus, both originating from acute otitis media, in which treatment was successful is detailed. That sulfanilamide played an important role in this cure will be borne out by the following facts.

REPORT OF A CASE

On Jan. 19, 1938, the patient, R. A., aged 7 years, was admitted to the ear, nose and throat service of Dr. Francis W. White, at the French Hospital. The history revealed that one week prior to admission there had been spontaneous rupture of the right ear drum. Drainage continued without any alarming symptoms until the day of admission to the hospital, at which time the patient was noticeably ill, with a temperature of 102 F., nausea, vomiting and tendeness of the mastoid. On examination, the right ear drum appeared extremely indurated and bulging, creamy-yellow pus discharging from a small perforation in the anterior inferior quadrant of the drum. A myringotomy was immediately performed, and the drainage then became more profuse. The next day the leukocyte count was 27,500, with 96 per cent polymorphonuclears, and 4 per cent lymphocytes. Roentgen examination of the mastoid gave negative results. The temperature rose to 105.5 F., and the tenderness in the mastoid increased. There was a 4 plus reaction for acetone in the urine. In view of the foregoing signs and symptoms, a simple mastoidectomy was performed. The operation revealed free pus and extensive destruction of the mastoid cells. The plates of the dura and sinus were apparently normal. A culture of material taken from the mastoid showed Str. haemolyticus. On the third postoperative day the patient began to show stiffness of the neck and a doubtful Kernig sign.

In view of these signs, the mastoid wound was reopened simply by separation of the edges, repacked and left wide open, and 10 cc. of prontosil [1] was given every four hours intramuscularly for four doses. On the fourth postoperative day the stiffness of the neck became definite and the Kernig and Babinski signs could be elicited. Spinal tap showed increased pressure and a cloudy fluid. Examination of the fluid showed a count of 4,200 polymorphonuclears, and later a pure culture of Str. haemolyticus was grown. At this time sulfanilamide therapy was instituted, as follows: 10 grains (0.65 Gm.) of sulfanilamide was given by mouth every four hours for eighteen doses and then 5 grains (0.32 Gm.) every four hours for twelve doses. During this course of treatment two transfusions of

1. The disodium salt of 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′, 6′-disulfonic acid.

200 cc. each were given. One of the remarkable features observed during this period of therapy was that within twenty-four hours the temperature dropped from 104 F. to normal and never rose to more than 101 F. subsequently, while the stiff neck and neurologic signs disappeared within three days. The spinal fluid taken on three successive days after the beginning of this therapy produced no positive culture and became clear of pus cells on the third day after the sulfanilamide therapy was begun. A tendency for the signs of meningeal irritation to recur was sufficient cause for the readministration of sulfanilamide on one occasion. The patient was discharged cured twenty days after operation.

This case was reported to the board of health, and the findings were confirmed by their diagnostician.

COMMENT

The high leukocyte count of 27,500 is noteworthy, as is also the cell count of the spinal fluid of 4,200 with a concomitant positive spinal culture of pure Str. haemolyticus. The tendency for the signs of meningeal irritation to recur was sufficient reason for the readministration of sulfanilamide.

CONCLUSIONS

1. Sulfanilamide is a powerful ally in combating infections with Str. haemolyticus.

2. The focus of the infection must be eradicated whenever possible.

3. The use of sulfanilamide should be discontinued after all signs of meningeal irritation have disappeared only if instructions are given to repeat the former doses on the reappearance of any of the former signs or symptoms, provided untoward symptoms do not appear before this time.

4. Surgical treatment comes first, then sulfanilamide therapy as an adjunct.

5. Without free drainage a sterile abscess may supervene, followed by destruction of bone and serious consequences.

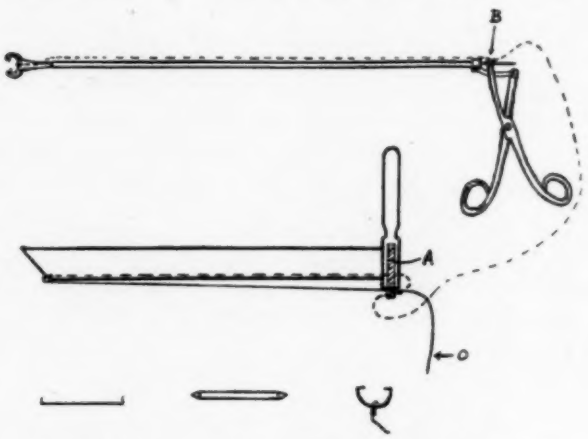
603 Bedford Avenue.

Clinical Notes

A USEFUL ADJUNCT TO THE POINT-SHEATHED METHOD OF REMOVING OPEN SAFETY PINS

KARL GRUPPE, M.D., UTICA, N. Y.

In the course of extraction of an open safety pin from the esophagus or tracheobronchial tree by the point-sheathed method, an arrest of progress is apt to occur as the head of the pin, being outside the endoscope, meets the cricopharyngeal pinchcock or the larynx, as the case may be. This is particularly apt to occur in the case of a small child, with a large pin or with a widely opened pin.



Method of applying traction to the head of a pin.

I am submitting herewith a method of applying traction to the head of the pin, a method which I believe has some advantages over the double forceps application technic, in that the connection is entirely flexible and hence does not interfere with the point-sheathing maneuver.

The method consists of clipping to the head ring of the pin a small metal clip to the center of which has been fastened a piece of braided silk or fishline (fig., lower part). The clip, made of thin copper or similar metal, is placed open in the rotation forceps. The fishline is then threaded down through the endoscope and an endoscope length pulled through and up to the handle on the outside, where this end and the line going through the instrument are held taut by a spring clip (*A*) on the head of the endoscope. A double forceps length of line continues from the inner piece and is attached to the clip held in the rotation forceps. This line is then fastened to the fixed thumbscrew of the forceps (*B*) to keep the slack from interfering with vision, and the setup is ready (fig., upper part).

The head of the pin is then visualized through the endoscope and the clip squeezed onto it under direct vision. The line is then released from the thumbscrew of the forceps and the forceps withdrawn. The entire length of line is then pulled down through the endoscope by traction on the outer piece (*O*) and the endoscope left clear. Several centimeters of slack should be left in the esophagus by partially withdrawing and replacing the endoscope before the loop is all drawn out, and a large coil of slack should be placed in the patient's mouth to prevent an accidental upward tugging on the line. The endoscope and pin are then free and ready for the point-sheathing maneuver.

News and Comment

AMERICAN BOARD OF OTOLARYNGOLOGY

An examination was held in Washington, D. C., Oct. 7 and 8, 1938, prior to the meeting of the American Academy of Ophthalmology and Otolaryngology. One hundred and twenty-nine candidates were examined, of whom ninety-seven were certificated.

The following examinations will be held during 1939: in St. Louis, May 12 and 13, prior to the meeting of the American Medical Association; in Chicago, October 6 and 7, preceding the meeting of the American Academy of Ophthalmology and Otolaryngology.

Application blanks may be obtained by candidates for certificates from the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha.

AMERICAN OTOLOGICAL SOCIETY

The annual meeting of the American Otological Society will be held on May 22 and 23, 1939, at the Westchester Country Club, Rye, N. Y.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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FUNCTIONAL EXAMINATION OF HEARING

ROBERT SONNENSCHEIN, M.D.
AND
NORMAN LESHIN, M.D.
CHICAGO

For a number of years we have noted that the majority of papers on functional testing of hearing have been by American authors. During the past year the preponderance of publications by our American confrères has been striking. There has been much attention to the subject of audiometers and hearing aids. The proper selection of aids to hearing is a "consummation devoutly to be wished," and perhaps this worthy objective is nearer than ever before.

The Council on Physical Therapy of the American Medical Association has authorized the following report on minimum requirements for acceptable audiometers:[1]

A. *Specifications.*—

1. Audiometers shall produce vibration at frequencies within hearing range (approximately 128 to 8,192 cycles, or higher, per second). They shall be equipped for testing both air and bone conduction.

2. Frequencies: (*a*) Continuous frequencies (sweep) from 128 to 8,192 (or higher, for example, to 16,000) cycles per second.

(*b*) Fixed frequencies from 128 to 8,129 cycles per second. If discrete frequency steps are provided, the tones shall be 128, 256, 512, 1,024, 2,048, 4,096, 8,192 cycles per second. Numerical annotations to be used designate pitch.

(*c*) The limits of tolerable frequency variation shall be not more than ± 5 per cent at all frequencies. Dials shall be marked so that frequencies may be identified readily.

3. Attenuation: Audiometers shall be calibrated in decibels, with 5 decibels per step or less. In no case should more than 5 decibel steps integrals be used. . . . Dials shall be easily read. The term "percentage hearing" shall not be used, but hearing losses shall be reported in decibels units loss.

The minimal range of intensity of the test tones above the normal threshold is stated.

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1. Tentative Minimum Requirements for Acceptable Audiometers, report of the Council on Physical Therapy, J. A. M. A. **109**:1812 (Nov. 27) 1937.

5. Wave Form: The purity of the tone in the air conduction receiver shall be such that the harmonics at any particular frequency shall be at a level not less than 40 dbs. below the fundamental tone or that other accessory noises . . . shall in anyway interfere with the test tone.

6. For a bone conduction receiver, the sensation level of the sound reaching the tympanum through the auditory meatus shall be at least 5 decibels below the level generated by bone conduction at all test frequencies as judged by a normal ear, when the bone conducteion receiver is placed on the mastoid region.

7. Power Supply: Either alternating or direct current, alternating-direct current or battery.

8. Ruggedness of construction to stand reasonable usage. Use of readily obtainable and replaceable parts.

9. Uniformity in calibration in decibels.

10. Advisability of the selection of a central, disinterested agency for reporting on physical characteristics of various audiometers.

11. Advisability of manufacturers assuring servicing.

B. *Audiogram or Auditory Chart.*—
(To be adopted later. . . .)

C. *Definition of Threshold of Hearing.*—The threshold of hearing is the audiometer setting corresponding to the lowest intensity at which the person being tested is able to indicate correctly more than half the time that he is hearing.

D. *Marketing and Advertising.*—Rules of the Council on Physical Therapy shall be adhered to by manufacturers of acceptable audiometers.

Fowler and Newhart [2] present the report of the representatives of the American Otological Society on the Committee for Hearing Aids and Audiometers of the Council on Physical Therapy of the American Medical Association and give their own comments. Fowler defines the cycle and decibel. He states that the range of intensity for 8192 cycles has been changed since the original report from 80 to 70 decibels. He suggests that complicated and tricky machines and charts be avoided. The determination of threshold of hearing as defined by the committee is peculiar to the audiometer and cannot be accomplished with anything like precision by tuning forks.

Newhart further states that the reason the committee has been unable to recommend as acceptable any of the several audiometers which have been submitted to it is the technical difficulty of formulating specifications for receivers of standardized performance. There are several instruments available, each having different features of real merit. The purchaser should buy that instrument which best serves his requirements, with the understanding that if the machine does not fulfil the specifications of the committee it can be exchanged or remodeled. They hope that this problem of a standardized receiver will soon be solved or

2. Fowler, E. P., and Newhart, H.: Report of the American Otological Society Representatives on the Committee for Hearing Aids and Audiometers of the Council on Physical Therapy of the American Medical Association, Laryngoscope **48**:519 (July) 1938.

that manufacturers can thus make acceptable audiometers. Newhart states the need for two types of additional pure tone audiometers, one especially designed for use in research dealing with the severely deafened person and the other a low cost instrument for rapidly detecting and accurately measuring loss of hearing in individuals or large groups by air conduction, having a range of only 128 to 8192 double vibrations and in no sense to be used for diagnostic purposes.

Ersner and his co-workers [3] investigated the performance of six of the most widely known and advertised audiometers and compared their calibrations with certain basic and fundamentally accepted standards. All the machines except one produced practically pure tones at all test frequencies. There was reasonable conformity among the audiometers in the intensity at the maximum indicated loss for at least the five lower tones but no such agreement at the position indicated for zero loss. Zero loss of hearing is shown by the universally accepted curve presented by Fletcher, representing the intensity of sounds at the average normal threshold of hearing. This factor was entirely overlooked by some of the manufacturers of audiometers. Only two instruments approached the proper intensities for zero loss, and these were in error by 17 decibels at 8192 cycles. The greatest errors in the audiometers tested, in addition to incorrect reference intensities, were in the attenuator or in its calibration. The intensity of the hum in all the audiometers except one was at least 60 decibels below the intensity of the test tone. The writers also give in detail some of the minor objections to each of the audiometers tested. They conclude that the errors found were due in general to lack of proper standardization and a central calibrating agency, to the attempt to build the machine to price rather than to standard of performance and to the failure of some manufacturers of audiometers to consider the known physiologic and auditory characteristics of the human ear.

They also state that the recent formulations fall short of providing standards and tolerances by which manufacturers may be guided, in that they do not define the basic and absolute standard.

Jones and Knudsen [4] emphasize the great service the otologist can render with audiometry and the prescribing of hearing aids. He is able with the audiometer to measure the exact amount of hearing both by air and by bone conduction and also to discover the type of impairment.

He can determine when a hearing aid should be prescribed, i. e., in cases of loss of hearing of more than 25 decibels (35 decibels at 4096

3. Ersner, M. S.; Podolsky, L., and Meyers, D.: Characteristics of Modern Electrically Operated Audiometers, Arch. Otolaryng. **26**:400 (Oct.) 1937.

4. Jones, I. H., and Knudsen, V. O.: What Audiometry Can Now Mean in Routine Practice, J. A. M. A. **111**:597 (Aug. 13) 1938.

cycles) throughout the range from 256 to 4096 cycles. The type of hearing aid, further, can be determined by the following rules:

If the loss of hearing is largely conductive and does not exceed the limits mentioned, a carbon type of aid should be recommended, because of its convenience and low cost. If the loss is much greater, more than 50 decibles throughout the range, for example, and the impairment is of a perceptive type, a high quality vacuum tube aid should be prescribed. If the impairment is primarily conductive, a bone conduction type of receiver is indicated; if the loss is primarily perceptive, the air conduction receiver should be used. When the two forms of deafness are combined, the choice of a hearing aid should be based on a thorough test of both types. If the loss of hearing is largely perceptive and much greater for the higher than for the lower frequencies, a high quality vacuum tube type of aid with appropriate emphasis of the high frequencies is indicated. When audiometric tests do not indicate a definite choice of instrument, careful speech tests should be made.

The writers bring out the shortcomings of the carbon type of hearing aid. The amplification is limited mainly to the frequencies from about 300 to 3,000 cycles and is not uniform. Undesirable nonlinear distortions also are present. The vacuum tube hearing aids, on the other hand, can amplify the entire range of frequencies for both speech and music without distortion. Any required degree of amplification remains constant over a long part of the life of the batteries; adventitious noise is low, and selective amplification can be obtained by using appropriate electrical filters. The main disadvantage of this type of aid is its lack of portability.

Knudsen and Jones emphasize the great necessity for audiometry both in the careful and accurate testing of hearing and in the prescribing of hearing aids. They emphasize also the importance of examining the vestibular mechanism. Persons practicing audiometry should know the basic facts of physical and physiologic acoustics and learn how to apply these facts to the technic of audiometry and the prescribing of hearing aids. They should also have a thorough knowledge of impairments of hearing and their relation to the entire human body. The acceptance by physicians and especially by otologists and collaborating physicists of the responsibility properly to familiarize themselves with this art will keep audiometry and the prescribing of aids in the hands of the medical profession, where it belongs.

H. Eschweiler[5] and Gerhard Steinberg,[6] in separate papers, both emanating from the university ear, nose and throat clinic of Prof. W.

5. Eschweiler, H.: Absolute Eichung eines aus käuflichen Apparaten selbst zusammengestellten Audiometers, Ztschr. f. Hals-, Nasen- u. Ohrenh. **43**:123, 1937.

6. Steinberg, G.: Praktische Erprobung des käuflichen Apparaten zusammengestellten Audiometers, Ztschr. f. Hals-, Nasen- u. Ohrenh. **43**:133, 1937.

Lange in Leipzig, Germany, discuss, respectively the absolute calibration of, and practical experience with, an audiometer recently constructed by Langenbeck from standard parts bought in the open market. Apparently this type of apparatus gave the observers satisfactory results. They discuss the advisability, however, of testing the calibration and other qualifications of the various parts used in the assembling of each audiometer.

Steinberg's conclusions may be summarized somewhat as follows: Audiometers constructed of parts normally found in industry fulfil the conditions for a practical instrument for audiometry. The author mentions the disadvantages of audiometry presented by Bayer a short time ago. The objections were: (1) the appliance is expensive; (2) its great weight and dependence on electric current make it difficult to bring the apparatus to the patient's bed; (3) there is no internationally accepted, accurate calibration; (4) the instrument needs constant watching and "caring for"; (5) the hearing curve obtained does not always show the type of impairment present, and (6) there is no clear relation between the curve and the understanding of speech.

Steinberg counters these objections by stating that the Langenbeck audiometer is moderate in price (900 gold marks, or about $350 at the present rate of exchange), especially when compared with the complete set of Edelmann tuning forks. The weight of the latter makes them also difficult to carry to the bedside. By using the normal curve for each particular audiometer, it is asserted, accurate results are obtained. Furthermore Steinberg states that the Langenbeck type of audiometer does not require any special care. The influence on the tests of age, sex, occupation, fatigue and other factors and the relation of the results to appreciation of speech are not definitely known with tuning forks any more than with an audiometer. The author emphasizes that no one method of examination is sufficient for accurately establishing the diagnosis, but that all available means should be employed, such as: (*a*) tests of hearing for whispered and conversational speech; (*b*) the Weber, Rinne and Schwabach tests with tuning forks, and (*c*) quantitative and qualitative determinations of hearing with the audiometer.

Ersner [7] emphasizes the importance of otologists' familiarizing themselves with the mechanics of hearing aids so that they may be better qualified to prescribe the proper aids for people with impaired hearing. He brings out the fact that this practical phase of otology is being neglected and salesmen who are not qualified in knowledge and whose one purpose is to make a sale are permitted to deliver this service to patients. He emphasizes the importance of special courses and lectures to explain the mechanics and value of hearing aids, which, moreover,

7. Ersner, M. S.: Is the Hearing Aid the Problem of the Otologist or the Layman? Ann. Otol., Rhin. & Laryng. **47**:212 (March) 1938.

should be standardized. The lay hearing aid technician may understand the physics of sound but is unfamiliar with the anatomy and physiology of the ear, the etiology and treatment of deafness and especially the psychology of the deaf and therefore is not properly prepared to render this service to persons with impaired hearing.

Phyllis Kerridge [8] has conducted a hearing aid clinic at the University College Hospital, London, since 1937. This was instituted because of the local need and as a result of an appeal from the National Institute of the Deaf. It provides a center for investigation and for demonstrations to medical students, physicians and teachers of the deaf. It combines medical science and an appreciation of human requirements in determining the need for and selecting the proper type of hearing aid. It is run in connection with the outpatient nose and throat clinic so that the services of an otologist are available for examination at all times. A history is taken to ascertain to what extent and under what conditions a hearing aid is necessary. The hearing is then tested carefully in a special sound-testing room for both air and bone conduction, and after study of the audiograms the proper hearing aid is prescribed. An impartial collection of hearing aids is available for testing. The patient is told which instrument is best for him, and a letter to the manufacturer is given him which enables him to try the instrument for seven days, after which he reports to the clinic what he hears and what objections or difficulties he has encountered. If the instrument is unsatisfactory, either another is tried or the patient is informed that at present none are available to suit him. The hospital also has the advantage of receiving a large discount for its patients, which is a great aid, especially when charitable organizations are assuming the financial obligation.

Hayden [9] gives briefly the fundamental features of audiometers and electrical hearing aids. By altering the construction of the transmitter and by changing the characteristics of the receiver, the sound of an electrical hearing device can be intensified within three divisions, low, medium and high, to fit approximately the loss of hearing shown on an audiogram. The electrical and mechanical characteristics of the transmitter, receiver and booster, when used in various combinations can produce hundreds of electrical hearing aids. The "response curve" of a hearing aid is obtained by passing the audiometer's calibrated tones into the hearing aid's transmitter, measuring the output with a decibel meter and plotting the over-all "tone characteristics." To determine the actual effect a hearing aid has on a patient's hearing, the audiometer's ear piece

8. Kerridge, P. M.: The Administration of a Hearing Aid Clinic, J. Laryng. & Otol. **53**:370 (June) 1938.

9. Hayden, A. A.: Audiometers and Hearing Aids, J. A. M. A. **110**:723 (March 5) 1938.

is attached to the transmitter of the hearing aid and the audiogram made. This is then plotted on the patient's original audiogram, and a graphic picture is seen of the hearing both with and without the hearing aid.

All but one of the four largest manufacturers of hearing aids fit their aids from the results obtained from an instrument called a master hearing aid. Hayden compares this with the trial frame used in ocular examinations. The other firm uses audiograms only, made in its own salesrooms. Two firms consider audiograms unnecessary and depend entirely on master hearing aids for their information. The fourth firm uses the audiogram of the otologist in addition to the master hearing aid.

To determine the real value of pure wave form, which has been considered necessary, especially by laboratory workers, and which increases the cost of construction considerably, the author used four different makes of audiometer to test 30 hard of hearing people. The four makes were the Sonotone, the Western Electric 6-A, the Western Electric 2-A and the Marvel. The Western Electric 2-A was taken as a standard of comparison because of its long established use. The Sonotone consistently showed the highest loss of hearing; the Marvel showed a medium but more erratic loss and the Western Electric 6-A the smallest loss. The average difference in decibels as compared with the 2-A audiometer was plus 5 decibels for the Sonotone, minus 3 decibels for the Marvel and minus 6 decibels for the Western Electric 6-A. All three audiometers were capable of measuring greater losses of hearing than the 2-A. Plus or minus 5 decibels was found to be the combined human error factor for both observer and patient. The author concludes that the audiometer is still the best means for testing acuity of hearing and that any one of the four used in the experiment was better than other methods used for testing. The lack of purity of sound did not introduce any serious errors. He also states that a quiet room is essential for testing and the need for a sound-proof room increases as the loss of hearing decreases.

Hayden [10] describes the salient features of the present hearing aids. The older methods of testing hearing do not give the information necessary for prescribing modern hearing aids, which is obtained by measuring hearing in terms of frequency at octave and preferably at octave letter steps and in terms of intensity, with the fundamental tone audiometer. Hayden tested four makes of audiometer clinically. The Western Electric 6-A requires frequent setting of the calibration to the zero beat because of the heterodyne beat circuit employed. The background noise is not sufficiently cut down, and the calibration of the dial should be more legible. Its wave form is good, and its sweep frequency is

10. Hayden, A. A.: Hearing Aids from Otologists' Audiograms, J. A. M. A. **111**:592 (Aug. 13) 1938.

really continuous. The Sonotone-Knudsen audiometer has excellent wave form throughout; its sweep frequency is above the conversation range, beginning at 2500 cycles per second. The Maico instrument has good wave form, but its "sweep check" is not actual continuous sweep frequency. The uniform zero level is commendable. The wave form of the Otologist Service audiometer is admittedly not pure below 1024 cycles per second. This appears of little clinical importance, especially in fitting hearing aids. Its continuous sweep frequency throughout is of great value in locating tone islands. Excellent features were found common to all.

The author suggested to the manufacturer the possibility of prescribing hearing aids from the patient's own audiogram. As a result of this, Otologist Service, Inc., was established. The Marvel audiometer was redesigned and rebuilt as the Otologist Service audiometer. Responses of the Sonotone hearing aids were made into Marvel's ingenious base-fitting charts on sheets of translucent paper that can be placed over audiograms for comparison. These charts and audiometers were made available to a small group of otologists. One thousand audiograms were made, and 300 of these were studied and "matched" and hearing aids assembled in the audioscope according to the prescription. The audiogram in all instances was invaluable in reducing the time and accompanying fatigue of the fitting.

A hearing aid is indicated when air conduction loss is greater than 25 decibels in each ear or not greater than 85 decibels in the better ear. When both ears are involved, if the difference in air conduction is greater than 20 decibels and the difference in bone conduction less than 10 decibels, one should fit the hearing aid to the ear with the greater loss for air conduction. If the difference in air conduction is less than 20 decibles and the difference in bone conduction greater than 10 decibels, one should fit the ear with the smaller loss for air conduction.

Selective amplification requires more clinical study and laboratory observation. The rule that "peaks of amplification" of the instrument should not be "matched" to peaks of better hearing in the audiogram is well established. Otologists must take greater interest in audiometers and hearing aids to prevent their exploitation by others not as well qualified to serve the hard of hearing. Otologist Service, Inc., offers a practical plan to aid otologists in prescribing hearing aids for the deaf.

Fowler [11] brings out the noticeable difference in the certainty of observations at the threshold in nerve and in obstructive deafness. The

11. Fowler, E. P.: The Use of Threshold and Louder Sounds in Clinical Diagnosis and the Prescribing of Hearing Aids: New Methods for Accurately Determining the Threshold for Bone Conduction, and for Measuring Tinnitus and Its Effects on Obstructive and Neural Deafness, Laryngoscope **48**:572 (Aug.) 1938.

ear with so-called nerve deafness responds with great certainty, whereas the one with uncomplicated conduction deafness responds with uncertainty to faint sounds, leaving some doubt whether they are heard or not. This difference, he explains, is due to the so-called recruitment phenomenon, previously described and reported, in cases of nerve deafness. To overcome misleading bone conduction measurements due to the inability to differentiate between hearing and feeling for frequencies near or below 500 and also to crossed audition, Fowler presents the following method: In the absence of nerve deafness or tinnitus, sounds heard by air conduction will sound approximately as loud as sounds heard by bone conduction at thresholds, and at given levels above the two thresholds. Variations in this phenomenon would indicate disturbances in bone conduction.

To measure the loudness of tinnitus, it is matched against the loudness of a similar frequency or frequencies put in the opposite ear, having no tinnitus. Fowler in discussing hearing aids mentions their one great fault, namely, that they lack the ability possessed by the normal ear to maintain automatically the normal proportion of sound with increasing distance. It is practically impossible with a light portable hearing aid to bring up the high frequencies to near normal loudness without overaccentuating the hearing for the low tones. Selective amplification in many instances is not sufficient without selective repression. Furthermore, the hearing aid does not automatically cut down the overpowering noise from nearby undesirable sounds, as an obstructive deafness will do naturally because of the impairment which acts as a masker of the noise. Some device which would act as a shock absorber, allowing full activation of the receiver up to a certain intensity and no more, should be incorporated in the hearing aid.

Hunter [12] describes a method in which tuning fork intensity can be interpreted in terms of decibels and gives in detail the development and final formula for this procedure. He used various methods also to determine the decrement of tuning forks and concludes that the decrement varies so much under different conditions that a careful technic would be necessary to get uniform results for a given observer. The technic, further, could not well be copied by another observer. From these observations he concludes that audiometers are the most dependable instruments in measuring the intensity of impairment of hearing. The rules given in this paper are given with the hope of improving the method of examination with tuning forks, but not with the idea that they can take the place of audiometers.

To see how the loss in decibels determined by tuning forks compares with that shown by the audiometer, 2 patients with conduction

12. Hunter, R. J.: Tuning Fork Tests in Terms of Decibels, Laryngoscope **48**:560 (Aug.) 1938.

deafness were tested with four audiometers of different makes and then with tuning forks. The loss in decibels indicated by the forks and also that obtained by ear were calculated by use of the noise level apparatus. No conclusions could be drawn because the results were so varied. When the decrement found by the ear was used the loss in decibels averaged from one-third to one-half the loss shown by the audiometers. When the decrement obtained by physical means was used, the loss was sometimes higher and sometimes lower than the reading of the audiometer.

Johnson [13] states that tuning forks should be an accurate and inexpensive means of distinguishing the type of deafness of a patient. This, however, requires a uniform procedure and method of interpretation. He offers a graph for the proper interpretation of the results of the tuning fork tests. Five forks, ranging from 128 to 2048 double vibrations, are used. The vibrating time in seconds is determined for each fork for both air and bone conduction, a pleximeter and a standard blow being used.

For transferring the findings to a graph each fork is assigned a fraction determined by using as the numerator the vibrating time of the fork heard the longest and as the denominator the average time for the invidual fork. In Johnson's forks the 512 double vibration fork had the longest vibrating period, thirty seconds by bone conduction. Thus a fork having a vibrating time of twenty seconds by bone conduction would be assigned the fraction 30/20 for that type of conduction. Air conduction fractions are assigned in the same way. The time for which the fork is heard by each type of conduction is multiplied by the appropriate fraction. In the instance given, twenty seconds multiplied by 30/20 gives thirty seconds, a value equal to the vibration time of the longest-sounding fork, so that the normal hearing time for the five forks when thus adjusted would be represented by a straight line on the graph. In each case the subject's hearing time for the forks, having been reduced to a common level by multiplication by the fractions established for the various forks, can be plotted, separate charts being used for air and for bone conduction. Johnson gives in detail the method and examples of its application. He states that its use does not mean elimination of the audiometer but offers an accurate means of checking its findings.

Wells [14] points out the fallacies of present hearing tests. The discrepancies of the ordinary voice and whisper tests arise from the noisy environment; the inability to mask the other ear entirely, the small

13. Johnson, F. A. H.: Interpretation of Results of Tuning Fork Test for Deafness, Arch. Otolaryng. **27**:304 (March) 1938.

14. Wells, W. A.: The Facts and Fallacies of Hearing Tests, Laryngoscope **48**:137 (Feb.) 1938.

spaces employed and the impossibility of standardizing the voice. He also calls attention to the point made by Knudsen and Jones, that the apparent increase in bone conduction in cases of conductive lesions is due to the noisy environment in which the test is made.

The objections frequently made to tuning forks are again mentioned. These are mainly the difficulty in standardizing the intial blow, the presence of overtones and the change in damping produced by various ways of grasping of the stem of the fork. He warns against generalizations concerning the reaction to the Rinne test in conductive and in perception deafness and gives an execption to the rule, such as a negative reaction in severe advanced nerve deafness. He suggests comparing the bone conduction of the patient with that of the examiner in doing the Schwabach test.

In discussing the audiometer, he states that it is the best instrument for scientific work and testing, in spite of its cost and of the fact that it is not as yet perfect. However, he emphasizes here also the necessity for testing in a quiet and if possible sound-proof environment. The disadvantage of the ear phone in that it measures bone as well as air conduction is stated, as well as the necessity for proper masking, especially in cases of complete or advanced unilateral deafness. Masking is always necessary if dependable results are to be had in testing each ear separately. Hearing tests, he states, are for two purposes: to ascertain the degree of loss and to determine the cause of the trouble. For the first he believes the audiometer and a sound-proof room are indispensable if accurate data are to be obtained. For the second, namely diagnosis, he believes that, in addition to the audiometer, the tuning forks still have a place. The magnesium forks made by the Riverbank Laboratories are mentioned; they can now be obtained with frequencies as high as 20,000 double vibrations. These for practical purposes will prove satisfactory in ascertaining the upper tonal limit. Wells states that if the fundamental facts of present tests are kept in mind and precautions taken to avoid the fallacies, a fund of helpful information can be gathered.

One of us (R. S.) has on a number of occasions suggested that the Schwabach test be made more objective by using a fork for which the average duration of vibration has been determined and seeing whether bone conduction is normal, lengthened or diminished. If the examiner compares his hearing with that of the patient, he must know definitely the exact status of his own bone conduction.

Fowler [15] demonstrates the fallacy of assuming that similar sounds appear alike to all ears with equal acuity of hearing as determined by

15. Fowler, E. P.: Measuring the Sensation of Loudness, Arch. Otolaryng. **26**:514 (Nov.) 1937.

the standard tests, that the loudness of a sound always corresponds to the number of decibels above minimum audibility and that the sensation of loudness may be predicated with confidence on the usual functional tests for hearing.

In patients with unilateral nerve deafness the difference in sensitivity of the two ears diminishes when the intensity of the tone is increased, so that with high intensities the loudness becomes equal for the two ears. This is due to the fact that the deaf ear sends nearly the same number of impulses to the acoustic centers as does the normal ear when the intensity of the tone is high. This phenomenon is a result of two known facts, the overlapping of the peripheral and central end arborizations of the cochlear fibers and the existence of a maximal stimulation for both nerve fibers and nerve cells.

This phenomenon may be utilized as an aid to diagnosis. If one introduces into the better ear loud sounds, 10, 20, 30 or more decibels in excess of the intensity required for minimum audibility in the poorer ear, and then introduces in the poorer ear the same frequency at an intensity which would balance the sound in the good ear, valuable information can be obtained. If a smaller number of decibels is required to bring up the hearing in the deafened ear to that of the better one, the difference in hearing is due to a nerve lesion. If, on the other hand, there is no recruitment or difference in hearing at the increased threshold in the poorer ear, the lesion is a conductive one. With a normal ear or an equal bilateral nerve lesion also, no differences will appear with changes of intensity. At all intensities, in cases of lesions interfering with conduction the same differences in hearing are maintained between the two ears.

E. Oppikofer [16] believes that the "shaking" test (masking) has been underestimated in most of the textbooks, even the most recent, and states that it is not even mentioned in some. So far as he is aware, in no book is it definitely asserted that for monaural testing with conversation the Bárány or similar appliances lead to absolutely useless findings and that the shaking test must finally replace such instruments.

The author states that Hennebert in 1914 proposed a method of masking similar to Wagner's but failed to give him priority for the shaking test, which he actually described in 1912.

In Oppikofer's clinic 400 persons were most carefully examined with a noise apparatus and with the finger-shaking method. He concludes that when one is testing with conversational speech a noise apparatus like that of Bárány gives useless data. The finger shaking of course does not affect the opposite ear.

16. Oppikofer, E.: Ueber den Wert der Schüttelausschaltung bei der Hörprüfung mit Umgangs- und mit Flüstersprache, Ztschr. f. Hals-, Nasen- u. Ohrenh. **43**:66 (Dec.) 1937.

It is admitted, as stated by Mauss some years ago, that the finger shaking will not mask very high-pitched tones. Oppikofer points out, however, that medium-pitched tones are masked. If, therefore, the small a-1 (435 double vibrations) fork and conversation are not heard when the finger-shaking masking is employed, the author states, unilateral deafness is demonstrated.

One of us (R. S.) [17] discussed this test of Wagner in his review of the literature which appeared in the ARCHIVES in January 1925. At that time he said:

> We believe that unless the finger of the examiner who manipulates the ear is covered with a sterile finger cot, it might be possible to produce an infection in the auditory meatus during vibratory manipulation. Instead of the shaking test, would not the rubbing of the auricle by the palm of the hand produce just as much of an effect as the noise apparatus?

Hulka [18] presents a new masking apparatus which is simple in construction and overcomes most of the disadvantages of other masking devices. The instrument consists of an acoustically insulated electric door buzzer and an air current expelled from a rubber balloon, which produces only a minimum of extraneous noise and about 60 to 65 decibels of effective complex masking tone. The noise is conducted to the ear by a stethescope-like device and is mixed in the receiver with the air current. The buzzer is enclosed in a container with acoustically insulated double walls. Control tests on subjects with normal ears and with unilateral deafness indicate satisfactory performance if used for the intensities and frequencies produced by the Western Electric 2-B audiometer. The three masking devices recommended by the American Otological Society are said to have serious defects. The telephone buzzer does not sufficiently mask the tones above 2000 double vibrations when used with the air conduction receiver and emits too much extraneous noise; in bone conduction tests it does not sufficiently eliminate the hearing of the masked ear in the speech frequencies (256, 512, 1024), and yet it interferes with the hearing of the tested ear if its loss in hearing is less than 40 sensation units. The door buzzer has the same disadvantages and in addition produces a larger amount of extraneous noise. The blowing of air against the drum head produces little extraneous noise and is satisfactory for testing with the air conduction receiver, but in the bone conduction tests the masking leaves rather high shadow curves.

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17. Sonnenschein, R.: The Functional Examination of Hearing: Annual Summary on the Problems of the Deaf, Papers Relating to Deaf-Mutism and Education of Deaf Children, Mechanical Devices for Hearing, Lip Reading, and So Forth, Arch. Otolaryng. **1**:89 (Jan.) 1925.

18. Hulka, J. H.: A Masker for Bone Conduction Tests, Ann. Otol., Rhin. & Laryng. **47**:153 (March) 1938.

Hulka presents audiographs of a subject with complete unilateral deafness, a subject with a deep congenital defect in hearing and 2 subjects with normal hearing to demonstrate the efficiency of his masking device. A full description and drawings for the construction of this simple apparatus accompany the paper. He also describes a method of standardizing the masker when parts have to be cleaned, adjusted or replaced.

Macfarlan [19] has found it useless to compare hearing of speech and of single tones, because they differ so clearly. The best way to test speech is by the use of speech itself as a stimulus. The phonograph has made it possible to reproduce the voice at an even level which can be repeated at various dates under comparatively identical testing conditions. There are certain factors of variability which should be understood and recognized. Speech sounds vary in frequency; no two words are acoustically equally well heard, because of accent and inflection and differences in the subject's familiarity with the words; vowel sounds are more easily recognized than consonants, which are more important in the interpretation of the word; fatigue and attention are large factors, and finally, dampening by outside noise or fleeting sounds is important. These factors prevent speech from being a perfect test but it still is the best one available.

Fletcher has stated that there is a difference of 100 decibels between barely audible speech and speech so loud it reaches the auditory-tactile sense. Any loss in decibels is equivalent to percentage loss of hearing. Macfarlan feels that one is on surer ground in reporting this loss in decibels than in expressing it as percentage loss.

The otologist should establish a level of hearing loss at which he considers it advisable to recommend the use of a hearing aid. This implies the testing of hearing to determine the loss in decibels. Speech comes through at cycles ranging roughly from 300 to 3000. Noise which interferes with and dampens hearing of speech comes in for the most part below 500 cycles. Hearing of speech is most affected by the hearing in the zone 1000 to 2000. The low tones are not very important, yet many aids amplify them. Phonographic testing facilitates the selection of a hearing aid. One can test the patient's need of hearing low tones and in this way receive information for selecting a proper aid. The phonograph is used with a control capable of varying the speed of the disk, with the attenuator slightly above the patient's decibel loss. The first test is made with the disk revolving at the standard rate, 78 revolutions per minute. The speed is then increased; the low tones fade, and the high tones become more conspicuous, and another test is made. The patient usually makes a better showing with the faster

19. Macfarlan, D.: The Hearing of Speech, Arch. Otolaryng. **27**:151 (Feb.) 1938.

record. Otologists have been much aided by this test in advising patients as to the selection of an aid. With the new crystal receivers and the crystal phonograph pick-up, peaking in the 1000 cycle zone is avoided, and frequencies up to 7000 and 8000 cycles are transmissible. It is now possible for the first time to amplify speech across its frequency range electrically without distortion. The testing of speech hearing with phonographic speech is an indispensable adjunct to the study of deafness and determines the degree of the disability with which the patient is most concerned.

Newhart [20] presents the significant relation of defective speech and impaired hearing in children and stresses the importance of early discovery of defects in hearing. Of the 50,000,000 children of school age in the United States, 3,000,000 are hard of hearing and 4,000,000 defective in speech. Subnormal hearing causes a substantial proportion of defects in speech, since articulate speech is mainly acquired by imitation through hearing. Extensive use of audiometers, particularly among school children, has disclosed an amazingly high incidence of deficiencies in hearing overlooked by the methods previously available.

Newhart points out the importance of a quiet environment for testing, of testing each ear separately and of testing the acuity for both air and bone conduction. Extensive tests have shown that a slight loss of hearing may cause retardation, defects in speech and inferiority complex and behavior problems. The real cause may not be apparent to either school teacher or parents. He quotes Russell to the effect that the incidence of defects in speech is eight times as great among those with a loss of hearing as among normally hearing persons.

New York and Pennsylvania have already passed laws for adequate hearing tests and correction of speech for their school children. Several other states have similar legislation under consideration.

Newhart believes the following conclusions are warranted: Every child with defective speech should have a hearing test with the 2-A audiometer or its equivalent. Persons working in the correction of speech should have a knowledge of the fundamental principles of testing the hearing with special reference to its relation to speech.

Departments of speech in universities should stress the importance of early correction of speech for hard of hearing children. They should encourage lip reading and periodic hearing tests as an important feature in the educational program of their respective states. Developments in electroacoustics and improvement in hearing aids can be of great value in advancing the science of correcting speech. An effective campaign of education among physicians, educators and parents is necessary for an earlier attainment of conservation of speech and of hearing.

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20. Newhart, H.: Hearing Deficiencies in Relation to Speech Defects, Laryngoscope **48**:129 (Feb.) 1938.

Newhart [21] stresses the importance of the early discovery of deficiencies in hearing in children to make possible more effective treatment of the diseases of the ear which cause them. In view of the fact that a large percentage of deficiencies in hearing can be prevented by the application of knowledge now available to every physician, it can be said that the ear, in proportion to its importance, is the most neglected organ of the body. Several factors are responsible for the poor results in this field of preventive medicine. The most important ones are the physiologic factor of safety, because of which a considerable loss of hearing may occur before the person is aware of the defect; the inadequacy of the older methods for testing acuity of hearing to disclose slight deficiencies, and the inaccessible anatomic location of the ear, a barrier to otologic research, diagnosis and effective treatment.

For effective testing of the hearing a proper environment is necessary to suppress extraneous noises; each ear should be tested separately and the threshold of hearing determined at different frequencies for both bone and air conduction. Regular periodic testing of the hearing of children of school age is the most effective measure on a large scale in the field of prevention of deafness. Audiometers of different types afford the most accurate and practical means of detecting and measuring impairment of hearing. The early detection of deficient hearing should be made a regular part of the school health program, in order to provide adequate educational care, to make possible early medical and surgical treatment and to safeguard the child for future life against a severely handicapping loss of hearing. There is an urgent need for the enactment of federal and state laws to make possible the incorporation of periodic audiometric tests in the public school health program. In cases in which the loss of hearing is greater than 40 decibels and the use of group or individual hearing devices is of great help to the child in interpreting articulate speech, these devices should be employed. The actual cost of regularly testing the school population with the audiometer is more than compensated by the saving effected through reducing retardation of the children. By using corrective treatment, instruction in lip reading and electrical hearing aids, retardation can be reduced by about 50 per cent, with a saving of at least $3,600 per thousand pupils.

Fowler [22] presents the results of hearing tests done before and after radical mastoidectomy in 54 cases. The audiogram curves were averaged for the frequencies most commonly used for hearing speech (256 to 4096). These showed that the operation did not benefit hearing in either ear. On the other hand, there was an average loss of hearing

21. Newhart, H.: The Early Detection and Treatment of Defective Hearing in Children, J. A. M. A. **109**:1620 (Nov. 13) 1937.

22. Fowler, E. P.: Hearing Before and After Radical Mastoidectomy, Arch. Otolaryng. **26**:387 (Oct.) 1937.

of 5 decibels both on the side of the operation and in the better ears. This average for 54 cases means a definite average loss and should not be considered a normal variation which could be absorbed or smoothed out in the average curves. Fowler makes a plea for proper treatment of acute inflammations to avoid the necessity of radical operative measures.

Bunch [23] undertook a study of traumatic deafness in the hope of throwing some light on this important clinical and industrial subject. He reviews the literature with special reference to boiler makers, persons exposed to gun fire, telephone operators, caisson workers and other patients with traumatic deafness. His subjects were examined with the 1-A Western Electric audiometer in a sound-proof room. He agrees with the opinion of the majority of the writers, namely, that bone conduction is diminished, that the response to the Rinne test is positive and that to the Weber test lateralized to the less involved side, that loss of acuity is greater for high-pitched tones, that tinnitus is not always present, that the tympanic membranes are usually intact, that deafness is most marked after excessive stimulation, after which there is a variable period of recovery, leaving a loss of hearing, which may be so slight that it is not noticed by the patient or discovered in routine testing, that the onset of deafness is so insidious that the person affected is often unaware of any defect in hearing and finally that men are more frequently affected than women.

Bunch's study, in addition, reveals certain characteristics which appear to be diagnostic. A loss of auditory acuity resulting from excessive stimulation by industrial noises is first evidenced by an abrupt dip or gap in the hearing range, usually near 4096 double vibrations. With continued stimulation this gap becomes deeper and broader, to include tones above and below this area. Eventually extremely high tones become inaudible, and the abrupt drop is between c-4 and c-5. At times this gap approaches normal at c-6 (8192 double vibrations) with the greatest dip at c-5 (4096 double vibrations). If the loss does not extend downward to include c-4 (2048 double vibrations) the patient may experience little loss for the spoken voice. However, if c-4 and c-3 are included, deafness to the spoken voice may be pronounced. If trauma results in rupture of the drum membrane, with subsequent suppurative otitis media, the hearing curves will be entirely different. All ears are not affected alike by the same traumatic noise. Explosive sounds or loud noises in a closed chamber have a greater traumatic effect than similar noises in the open. This warrants certain precautions, especially for men working in tunnels or caissons and for telephone

23. Bunch, C. C.: The Diagnosis of Occupational or Traumatic Deafness: An Historical and Audiometric Study, Laryngoscope **47**:615 (Sept.) 1937.

operators. The medicolegal and compensatory features of this condition are evident. Only a scientific study of the disability will make possible the proper rendering of justice.

Kafka [24] brings out the need for more careful and thorough testing of the hearing of the college student. Having followed the testing of students in various universities, he presents the methods used in a few of the important schools. The routine method employed at Cornell University is the watch test. In certain cases, when there is a question of deafness, the subject is tested with tuning forks, whisper and conversation, and coin tests are employed. At no time are sound-proof rooms used. In the routine entrance examination at Cornell, 2 to 3 per cent of the students are found to have a deficiency in hearing. At Yale the whisper and conversation tests are used. No sound proof accommodations are available. One hundred and fifty-three of 5,317 were found to have defective hearing. This group was further examined by the ear, nose and throat department to determine the degree of deficiency. At Princeton within the last few years the electric audiometer has been used in quiet rooms. The University of Michigan used no routine tests unless a history of impaired hearing is obtained. In the instances in which tests are made, both tuning forks and the audiometer are utilized. No figures of the incidence of deafness were obtained at this school.

Kafka believes that college students on entrance should be given rigid hearing tests by competent otologists in sound-proof rooms. All colleges should, further, have a uniform classification of the deficiencies in hearing found among the student body. Those with defective hearing should be treated. By these methods deficiencies will be discovered and treated early, to prevent, if possible a progressive chronic condition.

Gardner [25] reports the results of hearing tests which he conducted on the children of Boone County, Iowa. This county, the first in the state to have a large percentage of its school children tested, is situated in a rich agricultural region, where living conditions are good and the people are particularly health conscious. The 4-A audiometer was used to test all pupils in a quiet room. The hearing was considered defective if there was a loss of 6 decibels or more. A total of 3,529 pupils were tested, of whom 198 were found to have defects in hearing. One thousand, six hundred and eleven pupils of the consolidated schools in all grades were tested, 93, or 5.8 per cent, showing a deficiency. Of the 1,868 pupils of the Boone School tested, from the third grade through high school, 94, or 5 per cent, were deficient. Forty-nine pupils from

24. Kafka, M. M.: Deafness in the College Age, Laryngoscope **48**:65 (Jan.) 1938.

25. Gardner, W. H.: Hearing Tests in an Iowa County, Laryngoscope **48**: 69 (Jan.) 1938.

the kindergarten and first and second grades also were tested, and eleven per cent were found deficient.

Further study of the 198 children revealed 70 with a 6 decibel loss in at least one ear, 54 with a 9 decibel loss and 17 with a 12 decibel loss in the worse ear. Fifty-seven, or 28.8 per cent, had a loss of hearing in one ear that ranged from 15 to 57 decibels. Fifty of the 198, or almost 25 per cent, had a loss of hearing in both ears. Of the 50, 9 children had a loss of 15 decibels or more in the better ear. The educator is particularly interested in the group with bilateral impairment of hearing, to determine how many are permanently handicapped, so that they may be given a better opportunity to progress to the best of their abilities.

The incidence of deficiencies in the various grades varied but slightly. The elementary group had 5.5 per cent; the junior high school group, 5.4 per cent, and the senior high school group, 5.2 per cent. This shows the importance of testing high school groups as well as children in the elementary grades.

Of the 198, 92.4 per cent had histories of previous aural trouble. Fifty-four and five-tenths per cent reported earaches, 31.3 per cent running ears and 22.2 per cent noises in the ear. Forty-three per cent were found to have diseased tonsils and adenoids, and 14.6 per cent had wax occluding one or both external auditory canals. This survey should be of particular value to all medical and educational officials who are interested in handicapped school children.

Ciocco [26] presents a method for determining the minimum intensity of sound to be employed in auditory masking, which he defines as the minimum intensity needed for the elimination of perception of the sound of a tuning fork of 512 double vibrations held in the midline of the head. After hearing tests for both air and bone conduction were made, the results of the Weber test were recorded. If after repeated trials the subject reports that he does not hear the fork definitely in one or the other ear, the lowest intensity of the masker needed to lateralize hearing completely to the ear not being masked is then considered the minimum masking intensity. In cases in which the reaction to the Weber test is definitely lateralized, two questions must first be considered: whether or not there is a difference in acuity of hearing by air conduction between the two ears, and, if a difference is present, whether lateralization occurs to the better or to the poorer hearing ear. According to which condition existed, the following conventions were adopted: When lateralization occurred and no difference in acuity of hearing by air conduction existed, the minimal intensity for masking was deter-

26. Ciocco, A.: Audiometric Studies on School Children: The Use of Auditory Masking in Hearing Tests, Laryngoscope **48**:183 (March) 1938.

mined for the ear to which the sound was not lateralized, by means of the method described previously. The same intensity was then used in testing the opposite ear. When the response to the Weber test was lateralized to the better hearing ear, the same procedure was employed to determine the minimum intensity to apply for masking when testing the poorer hearing ear. The intensity introduced in this ear for masking when the better ear was being tested was established at 30 decibels higher than the threshold for air conduction of the better ear for 256, 512 and 1024 double vibrations. When the reaction to the Weber test was lateralized to the poorer hearing ear, the intensity introduced for masking the better ear was 30 decibels higher than in threshold for 256, 512 and 1024 double vibrations. The intensity to be introduced for masking the poorer ear in testing the better one was obtained in the manner previously described.

Auditory masking was employed in testing 300 children whose reaction to the Weber test was not lateralized. The following information was obtained: The average minimum intensity for masking was almost 60 decibels. Although the intensity required for masking varies considerably among different persons, there is a positive correlation between the two ears of the same person. The minimum intensity is not related either to the ear being masked or to the opposite ear. The differences between the minimum intensities for masking the two ears of the same person are not related to the differences in acuity of hearing between the two ears. Masking one ear raises the auditory threshold of the opposite ear for air conduction, the increase being proportionately greater than the increase in masking.

Ciocco will present further observations on the effect of masking in a later publication.

Suggit [27] investigated the effect on hearing of fluid introduced into the tympanic cavity by way of the eustachian tube and also the variation in hearing that occurs in otosclerosis over a long period when no treatment is being given. This was done in order to evaluate properly the work of Gray and the discussion it produced in 1936. Eleven patients with characteristic signs and symptoms of otosclerosis were treated with physiologic solution of sodium chloride introduced by way of the eustachian tube into the cavity of the middle ear. Three showed an improvement comparable to that in the series treated by intratympanic injection.

A series of similar patients, untreated, were observed over periods varying up to twenty-two months, and an average variation of about 10 decibels was found for the range of 256 to 2048 double vibrations. Actual examination of the figures given for the series treated by injec-

27. Suggit, S.: Variation in Hearing in Otosclerosis, J. Laryng. & Otol. **53**: 294 (May) 1938.

tion into the eustachian tube, the series treated by intratympanic injection and 3 patients with nerve deafness fails to place the improvement of hearing outside the limits of normal variation. No treatment of otosclerosis can be considered of value unless it can produce an improvement in hearing appreciably greater than 10 decibels over the greater part of the range 64 to 8192 double vibrations.

Firestone [28] has made audiograms of 109 pilots during the past two and a half years to determine the effects of prolonged flying on the auditory mechanism. All the pilots showed deterioration in bone conduction and admitted some impairment of hearing but boasted that they were able to detect the slightest noises or defective sounds in the motor long before less experienced copilots. The greatest loss occurs within the first three thousand hours of flying, and from then on the progress is markedly slowed down. Firestone ventures the opinion that the loss is probably due to the vibrations to which the auditory mechanism is subjected, which may produce changes in the region of the oval window akin to otosclerotic changes. He gives the name of aero-otosclerosis to this condition. He suggests that every pilot should have an audiogram recorded before embarking on his career. Possible steps toward preventing this condition would be to cushion the pilot, in an effort to reduce the intensity of the musculoskeletal vibrations, and to institute periods of rest from flying, with audiometric studies to determine whether such freedom from flying might lessen the injury to the auditory mechanism. Further study along these lines is certainly indicated.

4518 Woodlawn Avenue.

55 East Washington Street.

28. Firestone, C.: Bone Conduction in the Experienced Pilot and a Probable Interpretation, Laryngoscope **48**:168 (March) 1938.

Abstracts from Current Literature

Ear

Otogenic Streptococcic Meningitis. Abraham M. Litvak and Marjorie B. Klughertz, Arch. Pediat. **54**:714 (Dec.) 1937.

In recent years there has been an increase in the number of reported cases of recovery from streptococcic meningitis. Seventy-seven recoveries occurred within the last five years, as compared with 46 recoveries in the preceding thirty-one years. In 1937 recovery was recorded in 32 cases. In 15 of these sulfanilamide or a derivative was used.

The writers report the case of a 7½ year old girl who recovered completely from streptococcic meningitis of otitic origin. The spinal fluid showed hemolytic streptococci on smear and culture seventeen times. Mastoidectomy was performed, and concentrated scarlet fever antitoxin and autogenous blood serum were administered intrathecally. The patient was discharged after forty-nine days in the hospital. Examinations one and two months after discharge failed to reveal any residual signs or complications.

Orr, Buffalo. [Am. J. Dis. Child.]

Petrous Apex Suppuration Involving the Carotid Canal and Causing a Horner's Syndrome. M. C. Myerson, Laryngoscope **47**:345 (May) 1937.

Myerson shows that infections which spread from the middle ear to the petrous pyramid may localize in either the upper or the lower part of the apical area. Those which localize in the inferior portion of the apex will involve the bone surrounding the internal carotid artery. The canal which lodges the internal carotid artery in the petrous bone measures from 1.5 to 2 cm. in length and is anteroinferiorly placed in its relation to the cavity of the apex. When the infection involves the sympathetic nerve plexus surrounding the artery, Horner's syndrome results. The pupil of the eye on the same side is contracted, the palpebral fissure is narrowed and the size of the eye appears to be smaller. Myerson reports the clinical history of a man aged 44 who had bilateral pansinusitis, with more marked involvement of both maxillary sinuses. On the day of admission (Nov. 2, 1936) a Caldwell-Luc operation was performed on the left antrum. His postoperative course was uneventful until the seventh day, when an acute infection of the upper part of the respiratory tract developed. Six days later he complained of pain and fulness in both ears, suppuration in both middle ears subsequently developing, so that about three weeks after admission the ear drums were incised. His mastoid processes appeared to be involved simultaneously with the cavities of the middle ears. On December 7, it became necessary to perform a simple mastoidectomy on the left temporal bone. At operation, a perisinal abscess was found; there was no indication that a thrombus was present in the sinus. The right ear became dry and remained so for some days. In the further development the author stresses the following aspects: 1. A nystagmus toward the left was noted, which indicated a perilabyrinthine spread of the infection. 2. A concentric contracture of the visual fields was present, which led to the performance of a sphenoethmoidectomy. 3. A Horner's syndrome was present. 4. Deep-seated pain in the glenoid fossa occurred on moving the condyle of the mandible. 5. The apex and the area of the carotid canal were successfully drained by means of a cervical incision. 6. The labyrinth on the opposite side was found to be dead two days before the spinal fluid had a cell count of 70. 7. An erosion of the anterior surface directly above the internal auditory meatus was found leading into the cavity of the apex. 8. Neither pus nor spinal fluid was found on incision of the dura of the temporal lobe or over the basal cistern. Did the

meningitis spread from the area of involved dura, or was there an unrecognized abscess of the brain? It is frequently difficult to diagnose the premeningitic phase of an abscess of the brain. It is doubtful whether suppurative labyrinthitis of the other side caused the terminal meningitis; yet this must be considered as a possibility. The author concludes that earlier exposure of the temporal surface of the bone might have been better.

EDITOR'S ABSTRACT.

OTOMYCOSIS. J. CHINIARA, Ann. d'oto-laryng., June 1937, p. 498.

According to Chiniara, otomycosis caused by Sterigmatocystis nigra is common. The clinical picture is that of acute diffuse otitis of the external ear, with intense earache of the neuralgic type and painful adenopathies. The diagnosis is made by microscopic study of fragments of the growth eliminated during lavage. The author obtained satisfactory results from local instillations of a 3 per cent solution of copper sulfate in 5 of 6 patients treated. Administration of local instillations of silver nitrate failed in 2 other cases. Recovery of a patient from a grave condition was obtained by intravenous injection of a 1 per cent solution of copper sulfate. The injections were given every other day in amounts of 10 cc. each until six had been given. For patients for whom the local silver or copper treatment failed, the classic treatment of iodized alcohol controlled the condition. Eight cases are reported.

EDITOR'S ABSTRACT.

TOPOGRAPHIC SYNDROMES OF THE CENTRAL VESTIBULAR PATHWAYS IN MAN. J. A. BARRÉ, Rev. d'oto-neuro-opht. **15**:353 (May) 1937.

Barré accepts the otologists' definition of the vestibular system, using the term "peripheral" for the end organs and the part of the nerve lying within the temporal bone and the term "central" for the rest.

I. Syndromes of the vestibular nerve are classified as: syndrome of pressure due to tumor of the acoustic nerve, irritative syndrome of the vestibular nerve and syndrome of complete deficiency of the vestibular nerve.

A. The syndrome of compression is characterized by a sensation of pulsion, sometimes inclination, of the head toward the affected side, vertigo at times, spontaneous nystagmus to the sound side, deviation of one or both arms and slight deviation of the body toward the diseased side. Labyrinthine reactions of the affected side are absent with the caloric test, normal or reduced with rotation and normal with galvanism. The reaction of the sound side to the cold caloric test is horizontal nystagmus, which may or may not be convertible into rotatory nystagmus; with the warm caloric test there is horizontal nystagmus, transformable into rotatory nystagmus by inclination of the head, and with rotation and galvanism the reactions are normal.

B. A pure irritative vestibular syndrome has not yet been demonstrated. In cases of serous arachnoiditis of the internal auditory meatus, for example, signs of irritation (tinnitus, rapid diminution of hearing, violent vertigo and marked caloric hyporeflexia) are associated with signs of deficiency.

C. In the syndrome of complete deficiency there is total areflexia with all tests. Areflexia with the galvanic test indicates profound degeneration of the vestibular nerve.

II. As syndromes of the bulbar segments of the vestibular pathways, Barré discusses tumor of the cerebellopontile angle, syringobulbia, the unilateral bulbar syndrome and multiple sclerosis.

A. In cases of tumor of the angle, with compression of the bulb, lateropulsion is marked and is often supplanted by titubation and, finally, by falling. Later, vertigo is less frequent than in the initial stages. Nystagmus is toward the sound side, but sooner or later it changes in direction toward the diseased side, while the arms and the body continue to deviate in the original direction. Signs of nystagmus often vary with the direction of gaze. The caloric test causes no

reaction on the diseased side, except deviation at times of the trunk and the arms. The cold caloric test on the sound side often results in horizontal nystagmus, with or without rotatory transformation. With the warm caloric test on the sound ear, horizontal or horizontal-rotatory nystagmus to the sound side may be produced. The results of the rotation test are variable. The sense of rotation during turning is often preserved. With the galvanic test, reactions may sometimes be obtained from both sides.

B. In the irritative type of vestibular syndrome resulting from syringobulbia, the nystagmus is rotatory, but may be horizontal or mixed. The intensity is often marked and varies with change in gaze. It increases when the patient looks downward and in the direction of the quick component. Vertigo is not frequent. Lateropulsion is often observed but is soon compensated; sometimes a staggering gait persists. There is deviation of the arms to the same side as that of the trunk. Reactions to the vestibular tests are usually normal; the threshold of stimulation is often low, and spontaneous nystagmus is easily reversible. The lesions of syringobulbia produce, as it were, experimental section of the arciform fibers, which plays a dominant part in the genesis of rotatory nystagmus.

C. Vestibular disturbances in the unilateral bulbar syndrome of Wallenberg are of two types. In the first the nystagmus is rotatory, and in the second, horizontal. In the first type the nystagmus is usually toward the side opposite the lesion; it appears suddenly and becomes less intense as time progresses. It may change in form when the patient looks to the opposite side. In the second type, when the lesion is low, at the inferior part of the descending root of the eighth nerve, the nystagmus is rotatory and is produced by alterations in the domain of the posterior inferior cerebellar artery. When the lesion is higher, in the region of the lateral fossette of the bulb (Foix and Hillemand), the nystagmus is horizontal and behaves as does that of the first type. Spontaneous, pathologic rotatory nystagmus is indicative of an irritative central lesion; it seems not to depend on special excitation of the vertical canals. The transformation of provoked horizontal into rotatory nystagmus disappears in association with numerous lesions of the cerebral trunk, especially those located in the lower portion.

D. The striking feature of the vestibular disturbances associated with multiple sclerosis is the marked predominance of subjective and irritative phenomena over those of deficiency. Vestibular disturbances are sometimes the earliest to appear; it is particularly important to recognize them when they are unilateral. In early stages there is vestibular hyperexcitability, but in more advanced disease the threshold is normal in 60 per cent of cases.

III. Three syndromes of the pontile segment of the vestibular pathways are described: vestibular disturbances in the syndromes of Foville; a pontile vestibular syndrome without disturbance of ocular motility ("pure crossed vestibular dysreflexia"), and abolition of vestibulo-ocular reactions, with conservation of vestibulospinal reactions. In the first type, represented by a case reported by Van Gehuchten, there were paralysis of lateral movements of the eyes, facial paralysis, vertigo, nystagmus and deviation of the body, caused by a tuberculoma of the left half of the pontile tegmentum. Instrumental tests revealed marked diminution of vestibular excitability on the left side. In the second type the syndrome reproduced the findings observed by Leidler and others in animals after extirpation of a cerebral hemisphere. Barré called this syndrome pure crossed vestibular dysreflexia and reported a typical case in 1935. The patient had a lesion on the right side. The various tests produced nystagmus to the right only, and the slow phase of the nystagmus was always to the same side as the deviation of the arms and the body. Bard's assumption that there is a chiasm of the central vestibular pathways is substantiated by this case. The third type, characterized by abolition of vestibulo-ocular reactions with conservation of vestibulospinal reactions, is represented by a case of syphilitic arteritis, with bulbopontile complications and facial paralysis of the side opposite the lesion. There were no vestibular symp-

toms. With the cold and warm caloric tests no nystagmus was provoked, but reactional movements of the arms and the body were correct. The lesion of the bulbopontile tegmentum had sectioned the vestibulo-ocular fibers and spared the deiterospinal fibers, thus conserving all the reflexes below it. No vertigo was produced by the tests. Except for the absence of vertigo, the findings corresponded exactly to the schematic syndrome which Jones stated to be characteristic for a lesion of this region.

IV. Syndromes of the annexed bulbar and pontile regions are described.

A. Barré classifies the vestibular disturbances associated with tumors of the fourth ventricle as the global syndrome and the syndromes of the central region of the ventricle, the lateral recess and the inferior and superior angles. The central and global syndromes of the floor of the fourth ventricle are characterized by hypersensitive vestibular reactions to all tests. In the later stages there are diminution and eventual abolition of provoked vertigo and nystagmus, while provoked deviations are conserved and correct. Spontaneous vertigo and staggering are marked.

The syndrome of tumor of the lateral recess resembles that of tumor of the cerebellopontile angle, with the following differences: In cases of tumor of the lateral recess there are usually precocious vomiting and unilateral tinnitus with normal hearing and pain in the region of the fifth nerve on the same side. Vertigo may be absent, but there is lateropulsion to the diseased side, followed by staggering and falling. The results of instrumental tests are not known.

The vestibular syndrome of the bulbar triangle of the floor of the fourth ventricle is illustrated by a case in which there were complaints of hiccup, difficulty in swallowing, insomnia and complex nervous crises of the bulbocervical type. There were bilateral tinnitus with hypacusis, vertigo, lateropulsion, deviation of the body to one side and vertical nystagmus on looking downward. The vestibular tests were normal. There were no signs of hypertension. The vestibular syndrome of tumor of the superior angle of the floor of the fourth ventricle resembles that of tumors of the aqueduct and of the valve of Vieussens. There is no spontaneous nystagmus or lateropulsion, and objective reactions to instrumental tests are normal, with a low threshold. Early spontaneous vertigo of short duration, with absence of provoked vertigo to normal instrumental tests, is particularly associated with compression of the upper part of the fourth ventricle.

B. Vestibular disturbances in tumors of the cerebellum, according to Aubry and Lereboullet, may be divided according to the location of the tumor in the posterior three fourths or the anterior fourth of the cerebellum. Absence of vertigo and exaggeration of nystagmic reactions are characteristic of tumors of the posterior fossa. In cases of tumor of the vermis, marked disturbance of equilibrium, with correct past pointing, is often observed. Vestibular disharmony is contemporaneous with other signs of cerebellar involvement in cases of abscess of the cerebellum. With cerebellar tumors the sign of vestibular disharmony is frequently observed. While this disharmony signifies disturbance of the central vestibular pathways, Barré has always found it associated with disease of the cerebellum. He has observed it also in cases of lesion of the peduncular tegmen.

V. Syndromes of the peduncular portion of the vestibular pathways are modified by the various locations of the lesion in the mesencephalon. Typical observations are spontaneous vertical nystagmus, long duration of the provoked nystagmus, difference between the threshold for the slow and that for the rapid phase of nystagmus; nystagmus of different types and amplitudes in the two eyes, and retractory nystagmus (rare). Vestibular disturbances are often coincident with disturbance in ocular motility of nuclear, supranuclear or peripheral origin. Optokinetic nystagmus is frequently absent entirely or in one direction. Provoked rotatory nystagmus is often absent, sometimes in one eye only, as is inversion of nystagmus when the head is inclined anteriorly. In a large number of cases the vestibulospinal reactions are normal while the vestibulo-ocular reactions are dis-

turbed, but sometimes the former participate when one or both arms or the knee is deviated upward. Perhaps such instances represent a vestibuloequilibratory complex. The diverse phenomena observed in the syndrome of the peduncular region become clear when it is realized that they are manifestations of lesions involving the posterior longitudinal bundle or its fibers of distribution and the oculomotor pathways in their supranuclear, nuclear or peripheral segments.

In cases of tumor of the third ventricle, vestibular disturbances consist of vertical and horizontal nystagmus.

VI. Vestibular disturbances in disease of the pallidum have been discussed in connection with epidemic encephalitis lethargica. Muskens has assigned to the pallidum the role of a superior vestibular center.

VII. Vestibular disturbances associated with lesions of the frontal pole were described by Delmas-Marsalet. Barré suggests that phenomena belonging to the equilibratory apparatus should be differentiated from those belonging to the vestibular apparatus. He doubts whether the frontal pole exerts direct influence on the vestibular apparatus. The frontal type of past pointing arises from the pydramidal system.

VIII. Vestibular disturbances have been described in cases of tumor of the temporal lobe, but none in other diseases of the temporal lobe. An important characteristic is the great variability of the vestibular phenomena, such as spontaneous past pointing in patients with perfect equilibrium as well as in those with marked disequilibrium. Spontaneous past pointing of the type characteristic of lesion of the frontal or the temporal lobe does not exist. The thresholds for nystagmus are equal on the two sides and are usually normal. In almost all cases in which there is hyperexcitability, the onset is recent. Provoked vertigo is feeble or absent.

IX. Vestibular and equilibratory disturbances associated with tumor of the parietal lobe are characterized by absence of spontaneous nystagmus, frequent and, at times, violent disequilibrium consisting of retropulsion and lateropulsion, similar to that observed in the tumors of the frontal and the temporal region. The provoked nystagmus has a low threshold, large amplitude, high rapidity and long duration and is, in some cases, interrupted by a slow tonic movement that brings the eyes into a lateral position. Sometimes conjugate deviation of the head and the eyes is observed. Provoked vertigo is relatively slight. Optokinetic nystagmus may be irregular or may be absent in some direction.

DENNIS, San Diego, Calif. [ARCH. NEUROL. & PSYCHIAT.]

SYNDROME OF LABYRINTHINE GLAUCOMA. H. TILLÉ, Rev. d'oto-neuro-opht. **15**:577 (Nov.) 1937.

Tillé reports 3 cases of labyrinthine vertigo in patients aged 83, 60 and 45, respectively. The first patient had had chronic glaucoma for twenty years, latterly associated with vertigo, tinnitus and deafness of the senile type. Vertiginous attacks appeared in crises, and vestibular examination suggested dissociation of the caloric and rotatory reactions. The labyrinthine syndrome was probably related to hypertension of the labyrinth, comparable to that found in the eye in glaucoma. The second patient had attacks of vertigo and falling and a lowered upper tone limit. The labyrinths were hyperexcitable. There had been chronic glaucoma for three years in the right eye and for ten years in the left eye. The left eye was completely blind; ocular tension was 60 mm., and the fundus was not visible on account of edema of the lens and media; the right eye had a tension of 35 mm.; the visual field was retracted as far as the isopter of 15 degrees, and there was a lunated annular scotoma. In the third case the otitic symptoms were evidently caused by syphilis. Diffuse syphilitic cochleovestibular labyrinthitis, deafness for high tones, tonal islands and dissociation of the caloric and rotatory reactions were observed.

Three types of the syndrome are described: (1) senile deafness and visual difficulties resulting from insufficiency of the capillary circulation; (2) syphilitic labyrinthitis in young persons with glaucoma, and (3) labyrinthine glaucoma due to cold edema of the endolabyrinth. In a large number of cases Quincke's edema is associated with glaucoma, but the triad of glaucoma, labyrinthine disturbances and Quincke's edema has not been reported. Until means of measuring the tension of the endolymph and the auditory artery have been devised, it will not be possible satisfactorily to distinguish labyrinthine glaucoma. It is not possible to differentiate glaucoma and stasis in the labyrinth as it is in the eye.

DENNIS, San Diego, Calif. [ARCH. NEUROL. & PSYCHIAT.]

FUNCTIONAL ARCHITECTURE OF MASTOID PROCESS. H. LOEBELL, Acta oto-laryng. **25**:240 (May-June) 1937.

Loebell points out that the mastoid process shows many variations as regards its size and shape but that it is nevertheless possible to distinguish certain types. For practical purposes the author differentiates the large from the small mastoid process and, as regards form, the wide, usually hemispherical, more or less blister-like protruding type from the narrow, usually flat type. To be sure, there are also transitional types. The author describes studies on 20 mastoid processes from men and women of various ages. A table gives a survey of the size and shape of the 20 mastoid processes and also indicates whether the digastric fossa was deep or flat and what type of pneumatization existed. The mastoid processes were decalcified and then treated according to the split line method. The author describes and illustrates the chief directions of the split or cleft lines in the mastoid process and the relations of these lines to the muscles that are attached on the mastoid process. In studying the muscles, attention was given to their transverse section and to the direction of traction which they exert on the mastoid process. In summarizing his observations, the author emphasizes that the part of the skull protruding from the whole cranium is in every part firmly attached to the surrounding cranial bones. Protruding mastoid processes show curved split lines on the planum mastoideum and exhibit an extensive pneumatization. The dependence of a better pneumatization on the curved form of the mastoid process was shown by the examination of 20 craniums from the anatomic institute. The author's opinion of the predominating influence of the "drawing" action of the muscles on the size of the mastoid process and equally on the development of the cells also can be confirmed under pathologic conditions. The author proves it by reviewing the literature on cases of wryneck and by 4 personal observations.

EDITOR'S ABSTRACT.

Pharynx

FAUCIAL LESIONS OF PURPURA. E. WATSON-WILLIAMS, J. Laryng. & Otol. **53**:181 (March) 1938.

The faucial lesions of purpura are of great interest, especially in cases in which cutaneous manifestations are few or absent. They are probably not rare in purpura due to intoxication, particularly with drugs containing the benzene ring. Two cases in which neoarsphenamine was probably responsible for the development of purpuric manifestations are cited. A third case in which there were extensive purpuric lesions in the mouth and the pharynx is described. In this instance "aspro" (probably a form of acetylsalicylic acid) was the only drug which had been taken, but it is not specifically suggested as a probable factor in the development of the disease. In case 1, besides the faucial changes, there were a hemorrhagic ring in the esophagus, 33 cm. below the incisors, and a few small purple spots on both legs. In case 2 the evidence of the disease was manifested only in the fauces. In case 3, in which, besides the faucial lesions, the involve-

ment was most severe, there were a circinoid eruption on the neck and the face and a petechial rash on the chest. There is no record of the platelet count or of the bleeding time in any of the cases. Recovery was complete in each instance. A review of the classification of purpura by various authorities is appended.

LE JEUNE, New Orleans.

SEPTIC COMPLICATIONS AND SEPTICEMIA OF NASOPHARYNGOBUCCAL ORIGIN. A. LASKIEWICZ, Rev. de laryng. **59**:113 (Feb.) 1938.

The complications of a slight infection may be more serious than the initial lesion. A latent infection of the ear or nose may become virulent as a result of malnutrition, grip or trauma. The characteristic findings are a high temperature, chills, an enlarged spleen, an increase in the white cell count and a shift of the Schilling index to the left. The cardiac muscle is weakened; the pulse rate may be 120; the right side of the heart dilates; the sounds are muffled, and there may be murmurs due to endocarditis or pericardial friction. The mucous membranes are cyanosed; the skin is jaundiced; the tongue is dry, and the red count is decreased. Frequently the blood culture is positive. The tense capsule of the enlarged spleen causes pain on respiration. Pulmonary abscesses or purulent pleurisy may develop. Albumin, blood and casts in the urine show the disordered metabolism of the kidneys, and frequency of evacuation indicates intestinal involvement. Inflammation of the joints, periosteum and bone marrow, with erythematous petechial hemorrhages and chorioretinitis, renders the prognosis grave. The causative organisms are variable. The author believes that pus, wherever found, should be evacuated. He performs tonsillectomy during acute infections.

BATSON, Philadelphia.

Larynx

TUBERCULOUS STENOSIS OF THE TRACHEA. I. COLLEDGE, Ann. d'oto-laryng., June 1937, p. 519.

The points of interest in the case reported by Colledge are the following: (1) that the patient had suffered in the remote past from tuberculosis of the lung, the larynx and the pharynx, from which he had recovered; (2) that grave tuberculosis of the trachea alone developed, with the other structures of the respiratory tract normal, and (3) that resection of the trachea improved the patient's condition. He regained weight, a clear loud voice, tranquil sleep and a comfortable life for several months. Suddenly he was found dead in his bed with no signs of asphyxia. Necropsy was not performed. The author believes that death was caused by a cardiac crisis owing to the effort of the patient's heart resulting from cough, which was due to irritation of the trachea by the tube.

EDITOR'S ABSTRACT.

NONBACILLARY LARYNGEAL LESIONS IN TUBERCULOSIS. JUSTO-M. ALONSO, Rev. de laryng. **59**:270 (March) 1938.

The tuberculous patient may have all the laryngeal diseases of the nontuberculous. This fact is frequently disregarded. Some lesions have been regarded as incompatible with tuberculosis, and others have been confused with similar lesions of tuberculous origin. Isolated lesions in the larynx and those coexistent with tuberculosis are considered. Some lesions present a difficult clinical picture when they accompany generalized tuberculosis. Rare lesions are not presented.

1. *Simple Conditions.*—Inflammatory or ulcerative catarrh may be due to the constant irritation of the secretions and to the trauma of the cough. The symptoms are less chronic than when the condition is due to tuberculosis itself. There may be difficulty in breathing and in swallowing, and only the most minute examination

will enable one to make a differential diagnosis. Vocal rest and instillation of phenosalyl and sodium benzoate are useful in treatment.

Several forms of laryngeal lesion have caused considerable discussion.

(*a*) Monocorditis, when acute or chronic and not part of a general laryngeal process, the author feels is due to tuberculosis.

(*b*) Laryngeal pachyderma, rarely due to tuberculosis but representing a type of response, is especially seen in the fibrous type of tuberculosis.

(*c*) Nodular corditis in its true form is not tuberculous.

(*d*) Contact ulcer (Chevalier Jackson), the author feels, is commoner in tuberculosis than has been supposed and is ordinarily catarrhal in nature rather than specific.

Aphthous ulcers, due to the poor general condition and lessened vitality of severely ill patients, are similar to the ulcers which may appear elsewhere. Laryngeal and tracheal diphtheria is rare in the tuberculous, both because the pulmonary disease is found in adults and because of the relative isolation of the patients. The diagnosis is extremely difficult.

2. *Paresthesia, Spasm and Disorders of Motility.*—Occasionally patients present paresthesia in tuberculosis. They may have the sensation of a foreign body and other difficulties without an organic basis. These complaints are similar to those of the same condition associated with hysteria or chronic tonsillitis.

Spasm is rare in the adult and is generally due to the lesions of the nose or pharynx.

Modifications of motility, involving the function of either phonation or respiration and due to (1) neural paralysis, especially paralysis of the recurrent laryngeal nerve, (2) muscular paresis or (3) feebleness of the muscle without definite lesion, may occur either at the early stage of invasion or in the final period.

BATSON, Philadelphia.

FOREIGN BODIES IN RESPIRATORY TRACT. T. FALCÃO, An. de oto-rino-laring. **3**:106 (June) 1937.

Falcão has removed foreign bodies from the upper segments of the respiratory tract in 22 cases. In 10 of the cases the removal of the body was done by a pincet introduced through a bronchoscope under roentgen visualization. The diagnosis is done by roentgen examination of the neck and bronchial region. For removal of a foreign body from a bronchus the patient is placed in the dorsal position before the roentgen screen; the head is turned to the pathologic side; the bronchus is anesthetized through the bronchoscope, which can be introduced by the mouth, and the foreign body, under the roentgen control, is grasped with a pincet and easily removed.

EDITOR'S ABSTRACT.

Nose

MALIGNANT DISEASE OF NASAL ACCESSORY SINUSES, WITH REVIEW OF ELEVEN CASES. F. R. SPENCER and W. C. BLACK, Laryngoscope **48**:77 (Feb.) 1938.

All the cases (11) in which proved malignant growths in the sinuses were treated in the Colorado General (charity) Hospital during two and one fourth years through 1936 are reported. The practical considerations in rendering the best and the most humane treatment possible are given in detail, with a selection of pertinent opinions and findings from the literature.

In 7 cases the maxillary sinus was first involved; the orbit was invaded in 6, and the hard palate in 5. Exophthalmos was present in several at the time of admission. In 6 cases the condition was regarded as inoperable at the time of admission; 1 patient refused operation; 2 had Caldwell-Luc operations and 2 the Barnes operation. All except 2 received at least some irradiation, radium or

roentgen. In 3 cases the tumor was of undetermined type (no biopsy), and there were transitional cell carcinoma in 1, squamous cell carcinoma in 4, neurosarcoma in 1, plasmacytoma in 1 and rhabdomyoma in 1.

Follow-up showed 2 patients living, 4 known dead and 5 not traceable.

The authors conclude: ". . . early removal of the tumor by surgical diathermy, preceded on the operating table by a biopsy, the use of the cautery, radium and the Roentgen-ray have all helped to save human lives. Surgical removal of the superior maxilla is now rarely used." This relatively small series shows the variation in malignant tumors, the necessity of biopsy before the start of treatment and the variation in treatment according to the type of tumor. A bibliography is included.

WOOD, Newark.

CHOLESTEATOMA OF FRONTAL SINUS. A. GESHELIN, Acta oto-laryng. **25**:207 (May-June) 1937.

Geshelin reports the clinical history of a man aged 32 who was hospitalized on account of a pain in the right side of the forehead and a discharge of pus from the right nostril. The anamnesis revealed that about nine months previously the patient had been struck on the forehead. At that time he had been hospitalized for a month. Soon after his discharge, an accumulation of pus appeared under the skin of the forehead. Lancing was done twice, and after the second lancing a fistula formed, which had not yet healed at the time when the discharge of pus from the nose began. During the hospitalization described in the report Killian's operation was performed on the right frontal sinus. This intervention disclosed pus and a large cholesteatoma which completely filled the right frontal sinus. A review of the literature revealed records of only 9 cases of epidermoid of the frontal sinus. The author gives a survey of the literature and discusses the pathogenesis of cholesteatoma. The reported case is of interest on account of the connection between the cholesteatoma and the trauma.

EDITOR'S ABSTRACT.

Miscellaneous

DISORDERS OF SPEECH. JOHN A. GLASSBURY, Arch. Pediat. **54**:516 (Sept.) 1937.

The development and the physiologic apparatus of speech are reviewed. Normal speech is dependent on the proper functioning of five separate mechanisms: the motor, the vibrator, the articulator, the resonator and the controller. The motor is the lungs, which supply the breath. The vibrator is the vocal cords, the activity of which produces pitch. The resonators are the cavities of the throat, the mouth, the nasal sinuses and the body as a whole, all of which give quality. The articulators are the lips, the tongue, the uvula, the soft and the hard palate, the teeth and the lower jaw. The controller is the brain, which contains the centers that govern the entire mechanism of speech.

Disorders in speech may be classified under two general headings, those of defective phonation and those of stuttering or stammering:

Defective Phonation	*Stuttering (Stammering)*
1. Aphasia	1. Hesitation
2. Lisping	2. Reduplication
3. Nasality	3. Use of starters
4. Hoarseness	4. Inability to telephone
5. Falsetto voice	5. Inability to reply
6. Imperfect pronunciation	6. Inability to introduce
7. Perverted speech	7. Substitution of sounds
8. Retarded speech	8. Special disabilities
9. Foreign accent	9. Failure to sustain

The causes of delayed speech are considered under two headings, central and peripheral:

Central Causes	*Peripheral Causes*
1. Mental deficiency	1. Tumor of the tongue
2. Hydrocephalus	2. Adenoids
3. Amaurotic family idiocy	3. Abnormal uvula
4. Friedreich's ataxia	4. Cleft palate
5. Little's disease	5. Harelip
6. Bulbar paralysis	6. Imperfect dental arches
7. Cerebral hemorrhage	7. Defective hearing
8. Injury to the brain	8. Deformity of the jaw
9. Tumor of the brain	9. Intraoral adhesions
10. Chorea	10. Paralysis of the tongue
11. Epilepsy	11. Stenosis of the larynx
12. Hemiatrophy of the tongue	12. Enlarged tongue
13. Acute infection	13. Tongue-tie

Stammering and stuttering are curable, but too many patients are the prey of speech quacks. The treatment requires patience, application and faith. The author's rules of speech are: Before speaking think, relax and inhale. When speaking, never repeat, never go back and lengthen the vowel sounds. Speak slowly, quietly and calmly.

ORR, Buffalo. [AM. J. DIS. CHILD.]

THE STUTTER-TYPE CHILD: SPEECH INDEX OF NEUROTIC BEHAVIOR. J. S. GREENE, J. A. M. A. **109**:187 (July 17) 1937.

Fifty per cent of the patients with speech disorders suffered from dysphemia (stuttering). A stuttering child is a nervously agitated organism demonstrating high emotional tone and excitation, with a tendency toward quick interruption or inhibition. Other types of hesitating performances may be present. The result is usually a state of anxiety and social maladjustment. Stuttering is considered a physical symptom of psychic conflict. Since the child's difficulty is one of social adjustment, study of his case is directed toward integration and organization of his personality in order that he may become adjusted to home, school and social environment. The neuropathic diathesis is aggravated by parental tension and neuroticism and by lack of harmony in the preschool environment. The mother must be given an insight into child psychology and the importance of harmony in the home, so that the child may be properly guided. In the handling of the child the pediatrician plays an important role, and by applying a few principles of mental hygiene he may effect the adjustments.

BONAR, Salt Lake City. [AM. J. DIS. CHILD.]

THE DANGERS AND TECHNIQUE OF SPINAL PUNCTURE. J. J. KEEGAN, Nebraska M. J. **2**:285 (Aug. 7) 1937.

The first requisite for a spinal puncture is an adequate neurologic examination. The general physician should be reasonably well qualified to make such an examination, even to the use of the ophthalmoscope. Without a competent ophthalmoscopic examination a spinal puncture should never be done on a patient suspected of having a tumor of the brain. If optic papilledema is present the spinal puncture should be done only with the greatest precaution to prevent fatal herniation of the cerebellum through change in intracranial pressure. The best position is that in which the patient is prone on the edge of the bed with his head and knees approximated. An irrational or noncooperative patient must be restrained or given a general anesthetic. The best insurance against breaking of the needle is to use a new rustless steel needle. Repeated painful prodding of the

back has been responsible for discomfort in the back for many weeks. A local anesthetic may be used. The point of entry usually selected is at about the level of the crest of the ilium, and the local anesthetic need not go deeper than the skin. One should carefully seek an accurate estimation of the direction of the needle. Smaller needles are used than formerly. A 20 gage needle is large enough for routine use. The pressure of the spinal fluid should be estimated by a manometer. The fluid should not be withdrawn too rapidly, not faster than 1 drop per second. It should be subjected to a complete study. Cerebellar herniation into the foramen magnum is the most serious complication. The warning signals of this during puncture are a rather abrupt drop in the flow of the fluid and in the pressure, a complaint of pain in the back of the neck, difficulty in swallowing, cyanosis and cessation of respiration. The symptoms may not develop until a few hours after puncture and may then progress slowly over several hours. When any of these symptoms develop, several things should be done at once: Stop the puncture, elevate the foot of the bed, give a hypodermic injection of caffeine and an intravenous injection of 500 cc. of 50 per cent sucrose, the latter to dehydrate the brain and thus reduce intracranial pressure. In a person with a cerebellar tumor, all these measures are likely to fail and an emergency occipital trephining with tapping of a lateral ventricle or a suboccipital decompression may be necessary. Cisternal puncture cannot be recommended for routine application, but it has the advantage of preventing postpuncture headache.

HAMILTON, Omaha. [AM. J. DIS. CHILD.]

FOOD ALLERGENS: A STATISTICAL ANALYSIS OF FORTY-THREE CASES RELATIVE TO THE GENETIC CLASSIFICATION OF FOODS. ORVAL R. WITHERS, South. M. J. **30**:918 (Sept.) 1937.

A patient who is sensitive to one food in a botanic family has a 50 per cent chance of being sensitive to others in the same family. According to the statistics presented, the probability that sensitivity will exist to more than one member of a botanic family is stronger for some families than for others. It is pronounced for the family of beans and peas and is present to a lesser extent for those of apricots and peaches, of walnuts and pecans, of oranges and lemons, of watermelon and of cereals.

The symptoms produced in this series were predominantly those of allergy of the respiratory tract, namely, chronic vasomotor rhinitis and asthma.

Twenty-one per cent of the patients could tolerate certain foods cooked but not raw. Cabbage, apple and onion caused symptoms most frequently when eaten raw.

SCHLUTZ, Chicago. [AM. J. DIS. CHILD.]

THE PROPER CRITERIA FOR LIP READING RECOMMENDATION: MEDICAL AND EDUCATIONAL CONSIDERATIONS. MOSES L. BREITSTEIN and OLIVE WHILDIN, South. M. J. **30**:924 (Sept.) 1937.

After examinations and hearing tests have established the diagnosis of permanent or progressive deafness, the problem of lip reading must be immediately settled. Several questions must be answered before a decision can be reached: 1. Is the deafness monaural or binaural? 2. Does the loss of hearing lie in the range of speech? 3. Is the patient working up to the level of his ability in school? 4. Is he happily adjusted in his class, socially and physically? 5. Is his behavior natural and acceptable? 6. Is there any indication of deafness in his speech? The answers to these questions will determine the child's need for lip reading.

Follow-up and retesting in any case in which deafness is suspected are of greatest importance.

In cases in which the destructive process of purulent otitis media has not advanced too far, treatment, either local or operative, which has improved the local aural condition has likewise improved the hearing.

SCHLUTZ, Chicago. [AM. J. DIS. CHILD.]

ANAESTHESIA FOR HARE-LIP AND CLEFT OPERATIONS ON BABIES. P. AYRE, Brit. J. Surg. **25**:131 (July) 1937.

Ayre found that nitrogen monoxide-oxygen anesthesia was not suitable for infants. In the first place, anoxemia was caused by nitrogen monoxide and, second, it was extremely difficult to adjust the amount of rebreathing to the small proportions suitable for a baby. He devised the following method:

Briefly, the apparatus consists of a T piece which is connected by a short piece of rubber tubing and a Magill angle piece to a wide bore Magill rubber catheter previously inserted into the trachea. Through one limb of the T piece oxygen and ether vapor is delivered from a Boyle or other continuous flow apparatus. The other limb remains open to the outside air; for convenience, a short piece of tubing may be attached and allowed to hang down beneath the operating towels. A strand of fine gauze fixed with adhesive strapping close to the open end of the latter tubing will wave to and fro with the patient's respirations, thus serving as a useful indicator to the anesthetist.

The advantages of this technic are: 1. The baby inhales fresh air and oxygen under as nearly normal physiologic conditions as possible, as the system is open to the outside air, without the intervention of anything, such as a rebreathing bag. 2. The "dead space" is reduced to negligible proportions, while the continuous flow of oxygen at the rate of from 1.5 to 3 liters a minute flushes out the lungs and effectually prevents undue accumulation of the products of respiration.

SUMMERFELDT, Toronto, Canada. [AM. J. DIS. CHILD.]

DACRYOCYSTORHINOSTOMY. R. E. WRIGHT, Lancet **2**:250 (July 31) 1937.

As the otherwise excellent method of Dupuy-Dutemps proved too lengthy for the busy clinic in India, Wright sought a quicker and easier method. The new method is characterized by a small facial incision, and the steps are shown in diagrams with explanatory notes. The difficulty in this operation, and particularly in this method, is the suturing of the cut edges in the sac and in the nasal mucosa. Any fine, long needle holder and a large-eyed, fully curved needle measuring from 6 to 7 mm. from the point to the eye will serve. The procedure is made easier by the adoption of lateral hinged flaps. A straight eyeless needle from 3 to 5 mm. long (with suture material attached) is easiest to pass a point of suture in a small deep hole. The bone in the floor of the lacrimal groove of the sac is removed, and the hinge flaps are approximated and united by two points of suture. The anterior lip of the opening of the sac is then sutured to the periosteum of the crista. The cutaneous wound is sutured.

Wright says that this method is as nearly bloodless as the ordinary operation and is preferable to extirpation in conditions suited for dacryocystorhinostomy, e. g., a dilated sac in a middle-aged patient. A study of the diagrams will explain the method.

ARNOLD KNAPP, New York. [ARCH. OPHTH.]

MULTIPLE METASTASIZING MELANOMATA. NORMAN PATTERSON, Proc. Roy. Soc. Med. **30**:1393 (Sept.) 1937.

A patient had pigmented areas in the mouth, the pharynx, the larynx and elsewhere, as well as nodules on the left side of the neck. Microscopic diagnosis showed these nodules to be melanotic carcinoma. CAMPBELL, Philadelphia.

Directory of Otolaryngologic Societies *

FOREIGN

Collegium Oto-Rhino-Laryngologicum Amicitiæ Sacrum

President: Dr. Louis Ledoux, Brussels, Belgium.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg 143, Groningen, Netherlands.
Place: Brussels, Belgium. Time: September 1939.

Hungarian Otolaryngological Society

President: Dr. J. Treer, Bocskay-u. 9, Szeged.
Secretary: Dr. G. Kelemen, Reáltanoda-u. 9, Budapest.

Sociedad Rioplatense de Oto-Rhino-Laringología (Argentine Section)

President: Dr. Raul Becco, B. Mitre 1690, Buenos Aires.
Secretary: Dr. Juan Manuel Tato, Santa Fé 1171, Buenos Aires.

Société Française d'Oto-Rhino-Laryngologie

Secretary: Dr. Henri Flurin, 19 avenue Mac-Mahon, Paris (17°).

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. Leroy A. Schall, 270 Commonwealth Ave., Boston.
Place: St. Louis. Time: May 15-19, 1939.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 8-13, 1939.

American Bronchoscopic Society

President: Dr. John D. Kernan Jr., 120 E. 75th St., New York.
Secretary: Dr. Lyman Richards, 319 Longwood Ave., Boston.

American Laryngological Association

President: Dr. George B. Wood, 504 Physicians' Bldg., Philadelphia.
Secretary: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Place: Westchester Country Club, Rye, N. Y. Time: May 24-26, 1939.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Harold I. Lillie, Mayo Clinic, Rochester, Minn.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Chicago. Time: May 10-11, 1939.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

SECTIONS:

Eastern.—Chairman: Dr. Frank E. Kittredge, Masonic Temple, Nashua, N. H.
Place: Boston. Time: Jan. 11, 1939.
Southern.—Chairman: Dr. Francis E. LeJeune, Maison Blanche Bldg., New Orleans.
Place: New Orleans. Time: Jan. 14, 1939.
Middle.—Chairman: Dr. T. R. Gittins, Davidson Bldg., Sioux City, Iowa.
Place: Sioux City, Iowa. Time: Jan. 19-20, 1939.
Western.—Chairman: Dr. Frederic G. Sprowl, Medical Arts Bldg., Spokane, Wash.
Place: Spokane, Wash. Time: Jan. 29, 1939.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Isidore Friesner, 36 E. 73d St., New York.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.
Place: New York. Time: May 22-23, 1939.